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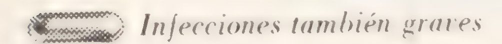
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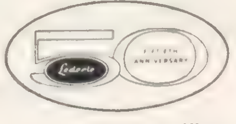
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DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 48 JULIO, 1956 No. 7

REHABILITATION IN GERIATRIC PRACTICE*

*RICHARD A. KERN, M.D.***

Neither the term "Rehabilitation" nor its concept is new to the physician. He has always had as his goal the return of his patient to full usefulness, not just the cure of his present illness. But some of us, especially those who are engaged in hospital practice, tend to lose sight of such complete rehabilitation, since our chief concern is the acute illness which brought the patient to the hospital, and when that is under control, we send the patient home to continue his convalescence.

World War II brought rehabilitation into sharp focus in the Armed Forces. There were cripples of all kinds who needed special training to compensate for the loss of a part or some function before they could return to civil life. Moreover, there were huge numbers of such cripples in every category. This wealth of experience produced many advances in the techniques and armamentarium of rehabilitation.

In April, 1944, on my return from sea duty in the Pacific Theatre to the U. S. Naval Hospital in Philadelphia, I became a member of its newly formed Rehabilitation Board, and later the Rehabilitation Officer and Chief of that Board. This gave me constant contact with many aspects of the extensive program in the Navy's chief rehabilitation center. During a 20-month period there passed through this service 159 blinded sailors, marines and coast guardsmen; over 900 amputees; over 3,000 deaf; many neuropsychiatric patients and a number of paraplegics. That experience was shared by many and could be translated in principle and often in detail to many aspects of clinical medicine in military hospitals.

The Basic Principles of Rehabilitation were well established in military practice. They can be stated as follows:

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* Presented on March 2, 1956, at San Juan in the Post Graduate Lecture Series of the Asociación Médica de Puerto Rico.

** Professor of Medicine - Temple University School of Medicine.

1. **Every patient who has been incapacitated by illness or by injury is in need of some rehabilitation.**
2. **This may be trifling in those whose incapacity is a mere matter of a few days.** Yet even the patient suffering from a severe cold has incurred a slight reduction in his ability to perform his daily tasks; this he regains quickly enough and without help or guidance, as a rule. But the patient with influenza is harder hit and takes longer to recuperate. The patient just over a severe attack of dengue knows what the Irishman means when he says, "it takes 3 months to get well after you recover." That patient may well need some planned help, even though it be largely a matter of reassurance.
3. **In others the need for rehabilitation is obvious,** whether there be an actual loss of a part or reduction of a function. Even the purely internal medical situations are clear enough: the diabetic must learn much about diet, urine analysis and insulin technique. The patient with Addisonian anemia must learn about his medical crutches. The cardiac cripple must know the limitations which his particular circulatory defect imposes (yet our failure to teach him this is all too often the cause for a later crack-up.)
4. **Rehabilitation of a patient begins on the day of his admission to hospital,** not in a week, or after the operation, or when the patient gets out of bed. The greatest trauma which the patient has suffered is often in the psyche, the hardest part of rehabilitation is mental readjustment: the sooner this is begun, the better and surer the result.
5. **Rehabilitation is a many faceted task.** The team that performs it includes many besides the medical specialist in whose purview falls the presenting lesion: the psychiatrist, the physiotherapist, the nurse, the chaplain, the social worker or Red Cross representative, and a host of special teachers. The teachers assume major significance when the patient is blind, or deaf, or an amputee. For the latter there are some two score procedures in daily living in which he must be trained, such as shaving, dressing and undressing, lighting a cigarette, dialing a number on the telephone, opening and closing a lock, driving a car, using tools. There must be learned new skills and handicrafts to replace old ones in gaining a livelihood. This may begin in the occupational therapy department with instruction in things not just meant to while away the time, such as basket making or leather work, but things useful in in-

dustry, such as assembling electronic devices, or watch repairing. Then the training can continue in on-the-job instruction in industry. Our blind patients became better darkroom technicians in X-ray departments than did seeing personnel.

6. **A Rehabilitation Officer must head up the team,** to direct the program and to coordinate its various activities.

What I have said of these principles has largely a military slant, and those in training were mostly young persons. But these principles are equally applicable to civilian general hospitals. But in these there has occurred a profound change in the age distribution of our patients. A couple of years ago Dr. Joseph Wearn pointed out that 20 years previously the average age of patients in his medical wards in Cleveland was 38, and that now it was 58. (Some wag wanted to know if he had kept his patients in hospital for 20 years). All of us have had the same experience. (If I see a patient under 30 on my wards, I wonder if I have strayed into the pediactric department). Today the beds in our general hospitals are largely filled with aging and aged patients, many of them past the acute stage of the illness that brought them to hospital, many in great need of rehabilitation.

What do we do with them? Let's be honest: do we rehabilitate them, or, as soon as the acute illness is over, do we try to send them home? Young house officers are certainly not too much interested in these older wrecks, and discharge them to their homes (if the family will take them), or to a nursing home, if one be available. But the oldest and most incapacitated, whom nobody wants? At long last, when a bed becomes available, they are transferred to some home for the aged, or a comparable municipal institution, where the bodily needs of the patient are met, but very little else, and where certainly no thought is given to rehabilitation. There they wait: to die. Yet before they are so transferred, we are "stuck" with them for weeks or even months, during which so much might be done, if we were so minded.

Today, rehabilitation is being pointed more and more toward the aged. This is chiefly because there are so many of them. In 1850, only 2.6% of the population of the United States had reached the age of 65. In 1950, there were 8% over 65, and by 1980 these oldsters will comprise 14.5% of the population. They are increasing at a rate five times as fast as the rest of the population. Now, of those over 65, only 5% are financially independent, 25% are still at work, and 70% are dependent wholly or in part on the rest of the people. What is more 2% of those over 65 are incapacitated by illness at any time, and of those over 80, no less than

25% are always on the sick list. This poses a tremendous and rapidly growing economic problem. Today those over 65 comprise 8 per cent of the population, but they are 11% of those over 18. That means there are 8 workers between 18 and 65 to 1 person over 65. In 1980, those figures will be 14.5% of the whole population, 20% of those over 18 and 4 workers between 18 and 65 for every oldster. Moreover, these are **average** figures for the whole country: in some areas the aged are more numerous by reason of their migration.

In 1941, when I first addressed you on this subject,[1] The Care of Aged, I must admit I was reminded of the Patron Saint of this city, San Juan Bautista: a voice crying in the wilderness, for the aged in your midst at the time were definitely less numerous than on the mainland. But in the last decade the picture has been changing rapidly. The percentage of aged has increased not only by reason of their continued survival from infectious diseases, but through the effects of migration. For the first time in many years the natural increase of your population has been offset by the processes of migration. Those who are going to the mainland are overwhelming the young: the old are mostly staying here. Moreover, I predict that in time those who left the island will be coming back when their aging bones in northern winters yearn for the warm sun of their youth.

There is another reason for a quickening interest in geriatric rehabilitation: an increasing number of aging physicians. The younger ones are not so moved by the problem of the aged: why go to so much trouble to gain a few more effective years? But the nearer one is to that age bracket, the more sympathetic becomes one's viewpoint.

So it is that there now is an increasing number of local islets of endeavor in applying the principles of rehabilitation to the aged, with a growing experience and a growing literature. Each day is improving the outlook for the aged to continue to be self sufficient as far and as long as may be, and, best of all, to be the happier for it; an outlook that applies not only to our aging patients, but sooner or later for all of us; indeed, mighty soon for me and even some of you.

The greatest need in my hospital and in yours right now is a Rehabilitation Service. I am safe in saying that, because the odds are great, that most hospitals now have no such service. For example, in Pennsylvania, Martucci[7] reports in February, 1956, that of 356 hospitals only 52 have a rehabilitation department, only 36 with a physician in charge. Others may lay claim to some rehabilitation attempts, but these are more likely to be in the nature

of elementary physiotherapy, laudable, but limited in scope, occasionally supplemented by so-called occupational therapy, occupying the patient's time but rarely leading to a gainful occupation.

The prime need of such a rehabilitation service is **a physician trained in the field.** (They are rare: of the 11,892 physicians in Pennsylvania, only 29 are certified in physical medicine and rehabilitation). In his absence, the next best choice is an interested physician, willing to give the assignment the necessary time, to build up a staff of technical assistants, to set up a consultation service to take care of referrals both in the hospital and in the outpatient department. (Krusen(5) reports that even now 1 in every 8 patients in the Mayo Clinic is referred to his rehabilitation service). In the absence of a chief of rehabilitation, each one of us, especially if we are heads of departments, has it on his conscience and responsibility to do something about the rehabilitation of our patients, and to train our students, interns, residents and staff members to develop that viewpoint.

It is not my intention to try to survey the field of rehabilitation this evening. That would take as many days as I have minutes at my disposal. But I would like to stress some examples of specific disorders and handicaps encountered in our old patients, and to show what may be accomplished in their rehabilitation.

Perhaps the commonest, the most disabling, and unfortunately the least-well-managed are the **cerebral vascular accidents.** Here let me stress that the major damage which the patient has suffered is not the loss of power of an arm, or a leg, or even of speech, but the break of communication between him and the everyday world of which, up to the minute of the accident, he was an active part, and from which he now suddenly feels himself completely and permanently isolated. His greatest trauma is a psychic one, and this needs the first and immediate attention of physician, nurse, family and all who have anything to do with him. We must maintain at all costs the bridge of communication between the patient and his own world.

But what if the bridge is completely broken? What if the patient awakens from his coma with an aphasia? He thinks, hears, sees, feels: but he cannot speak, answer or make known his simplest wants. His mental anguish must be the extreme of suffering. What can be done about it?

I am sure that all of you in early years have read the great work of Alexander Dumas: **The Count of Monte Cristo.** Then you must remember how the Count discovered the secret of the archcriminal by managing to communicate with the speechless paralytic victim who could only move his eyes. Yet the Count was able to

work out a system of signals that with time and patience brought all to light. Dumas there recorded a basic technique of rehabilitation that we for the most part have lacked the astuteness to exploit.

In 1943, Dr Hamilton Cameron[1] of New York City had a coronary thrombus, followed by cerebral embolism with right hemiplegia and complete aphasia. It was two and a half years before he was again able to speak. Yet in the first 4 weeks, while in hospital, he devised a series of 20 hand signals with which he was able to make known his simple wants. In the years since then he has published in many journals his "hand-talking chart," that has helped to open the "Door of Silence" for many of these unfortunate people. It took the doctor 4 weeks to work out for himself what we can now do for our patients in a day or two.

Most hemiplegics can be trained to be independent at home even if not a single muscle regains power. Moreover, the return of power is aided by activity and such improvement can be hoped for during two full years after the stroke. The phases of training, as outlined by Mahoney, Barthel and Callahan,[6] include: (1) self-care and ambulation, (2) the recovery of all possible power and range of motion, and (3) training in coordination, balance and endurance. **Motivation is the most important thing, and therefore comes first.** This means contact by a sympathetic and cheerful approach, but stressing independence, and not letting him enjoy invalidism. Early treatment also prevents contractures and weakness.

Rehabilitation begins as soon as the patient can hear, understand, is in a stabilized condition, and can obey simple commands. Progression of rehabilitation, as Mahoney and her associates point out, is governed by the limits imposed by mind, balance, circulation and effects of dependency. Physiotherapy begins at the bed side, with passive motion of an arm, a leg, then active motion when possible. Speech therapy begins with signs, as above noted, and progresses by repeated trials. In all of this, one watches the patient for signs of fatigue or stress, such as flushed face, increasing pulse and respirations.

The patient is first taught how to change his position in bed, then how to get in and out of bed. He learns to sit on the edge of the bed, and to preserve his balance while sitting. Next he is taught to stand by the side of the bed, then how to get in and out of a chair.

He learns how to snap and to open the fasteners of his pajamas, and how to put on and remove the pajama coat. He is taught to put on slippers and a robe with a belt. He learns to feed himself and to attend to his personal toilet. Walking, the use of a wheel

chair, how to negotiate a shower on a stool, getting into and out of cars, how to get up and down steps with a handrail are successive steps in training. For further details the reader is referred to the excellent article by Mahoney, Barthel and Callahan, upon which I have freely drawn. There is a long list of every day skills to be recaptured. Once the person has learned a skill, no one should help him.

The final stage includes the fitting of braces to help walking and stability and then occupational therapy. Here the possibilities are limited only by the intelligence and previous knowledge of the patient. They should always include the use of skills the patient is known to have. Let me remind you that Pasteur did much of his best work, including that on rabies, years after his hemiplegia. But one need look no farther than this very city and this Association for a notable example: your own Dr. Pablo Morales Otero, whose facile pen continues to perform a signal service in interpreting medical science to the laity.

Parkinsonism is a cause of major disability. That it need not fetter a brilliant mind, you have all seen amply demonstrated. Much can be accomplished by means of drugs. In addition to the older ones: belladonna, hyoscine and strammonium, we now have the antihistaminics, such as benadryl and pyribenzamine which commonly exert a sedative effect and which at times work best in combination with belladonna. Then there are various new synthetics, such as trihexyphenidyl and mephenesin. They again may be tried singly or in combination with other drugs so as to strike the most efficient therapy.

In every case there is need for training the patient in ways and means to protect him against injury, and he can often be helped in recapturing some basic skills.

Nor should we neglect more direct measures in patients who are seriously incapacitated. The improvement in both tremor and rigidity which has followed occlusion of the anterior choroidal artery has led to the use of measures aimed to destroy the mesial portion of the globus pallidus. (This structure exerts an excitatory influence on motor activity initiated by the cortex and mediated by the pyramidal tract). Recently, Cooper and Poloukhine(2) in New York City have advocated the injection of 0.75 ml. of absolute alcohol under local anesthesia, the patient being in sitting position; the injection proceeds very slowly and ends when tremor and rigidity are relieved. They report on a series of 70 cases, with immediate good results in 80%, lasting good results in 70%, 2 deaths and 1 hemiplegia. Their successes have been achieved in elderly arteriosclerotic patients as well as in younger cases of inflammatory

origin. There has recently been seen in this city a man who had been a helpless invalid from Parkinsonism and whom such a **chemopallidectomy** had restored to virtually a normal state.

However, this approach is not without danger, as the record shows, and this is because of the obvious difficulty in exact localization of the site of injection and in controlling the extent of tissue destruction. I therefore call your attention to the more accurate approach by Spiegel and Wycis.[16] [17] By means of an ingenious guiding apparatus, the stereoencephalotome, which they have devised, they are able to localize the tip of their electrode with a high degree of accuracy, and then to produce by electrolysis or electrocoagulation a very small circumscribed lesion with minimal injury to overlying cerebral structures and with less danger to adjacent subcortical tissue than an injection procedure would entail. This, too, is done under local anesthesia so as to have the patient's cooperation and reaction at every moment and stage of the operation.

By these several approaches, we may in a high percentage of our patients be able to show that Parkinsonism is a reversible and not a hopeless condition.

From cerebral vascular accidents and Parkinsonism it is but a step to the **psychiatric problems** of the aged. I shall not concern myself with the psychoses of the aged, frequent though they are, and for which much can be done by newer methods of treatment, including drugs, such as reserpine and chlorpromazine, and electric shock therapy. I wish rather to refer to the more subtle but almot universally present problems of maladjustment of the aged to those around them. So long as a man can work and produce, he maintains his place in the social structure and he has no psychic problems on that basis. But when he is no longer able to work, or has been forced to retire, then these problems begin and grow apace. They have a two-fold origin, as Wilson[13] has recently emphasized. They begin with the attitude of those around him who tend to ostracize him as one no longer able to meet the standards of the group. He in turn, whether he fights back or voluntarily withdraws, increases the degree of his segregation. The effect upon his emotions is devastating, what with feelings of failure, insecurity, of being a burden and unwanted. Illness accentuates all of this, and hospitalization is often the greatest blow to what is left of hope and self-esteem. When the transfer is finally made from hospital to so-called nursing home, or home for the aged, or county home and poor house, where visits from the family taper off and cease, the oldster knows he is forsaken and his withdrawal becomes complete.

Yet in these apparently hopeless persons some amazingly good

results can be achieved, if one can by kind and persistent attention rekindle in such a patient an interest in those about him. I recall a recent patient, a woman in her eighties, who was transferred to our ward in an apparently terminal coma. She was wholly unresponsive and during two entire weeks lay with her face to the wall. Yet various tests showed no evidence of serious physicochemical abnormality. Then one day she responded to the pleasant approach of a young nurse who was brushing her hair. Within days she was up in a chair and making friends with others on the ward.

And now I call your attention to a report on this subject by Munger and Jarrett.[illegible] In 1946, there was begun a rehabilitation program in the Allegheny County Home of Pittsburgh, an institution with 1,800 patients, all of them indigent and chronically ill. To show what might be accomplished, the Rehabilitation Service asked to be given the 12 worst cases to work on. They proved to be 12 such completely withdrawn individuals, who had been bedfast for from 2½ to 6 years, speechless, snarling and spitting at those who disturbed them as they lay in their own filth. The first move was to put them in a round room, their beds arranged in a circle, to make them conscious of one another. Then came a patient persistent effort to arouse their interest. Physiotherapy in bed and exercise by ropes and pulleys brought new strength to enfeebled muscles. The final result: 4 were sufficiently well to be received back into their own homes, 5 more became ambulatory and were transferred to another department for further re-training, and only 3 remained bed-fast. That same rehabilitation service has been able to discharge 70% of the amputees in the hospital to their homes and 25% to actual employment, 60% of arthritics to home and 17% to employment, 61% of patients with fractured femurs to home and 15% to occupation, and 40% of hemiplegics to their home as able to care for themselves.

Our **cardiac patients** present the commonest challenge, both in the old and the not so old, a challenge which, I fear, we all too often do not adequately meet. Our errors are in both directions, too little or too much concern with the future. The patients with myocardial failure are carefully nursed back to compensation which at best is backed up by a sharply limited cardiac reserve. Yet we pay no attention to their nervous occupation and way of life, that may involve heavy labor and the expenditure of undue energy in climbing many flights of steps in home or factory or subway. The unwarned patient returns to these circumstances, only to break down promptly and be worse off than before. At other times we are too cautious about lesser cardiac troubles or about those from

which patients can make a good recovery. I have seen patients with only extrasystoles made cardiac invalids by overzealous physicians. How well our patients with coronary occlusion can do is being amply demonstrated in high places. I hasten on to other subjects, lest the discussion become political. I do wish to remind you that aged hypertensive patients should not be treated too vigorously with drugs that lower blood pressure, lest they develop thromboses.

The **rehabilitation of arthritis** offers plenty of opportunity for imagination and ingenuity in devising ways and means to help them overcome their handicaps. I have a friend who has a hip ankylosed in the straight position, so that he is unable to sit, or to stoop and pick things up. His spine is also nearly rigid so that he cannot turn his head. On every wall of his home are extension tongs with which to pick things up. His chair at table is in a recess in the floor (a slightly lowered elevator), so that, although he is virtually erect, his face is on the same level as the faces of the others at the table. His station wagon at the driver's seat has a lowered floor, so that he can drive erect, as does the milkman. At his bed and by his library chair, a series of properly placed mirrors bring into view each door, window and chair in the room. I could mention many other gadgets. The significant thing is that he is the active head of a large manufacturing business.

There are many aged **leg-amputees**, and many of these have been allowed to lapse into a helpless bed or wheel chair existence, or at best into complete dependence upon crutches, rather than providing them with prostheses. This is of course the item of expense, but this is now less than it used to be, and the efficiency of the artificial limb much improved, thanks to newer devices and to mass-production of many parts. Success with a prosthesis is due first of all to the enthusiasm and the skill of the rehabilitation staff in teaching the patient how to use it. You don't buy a boy a violin and expect him to learn to play it without instruction. Every one must be taught how to walk with an artificial leg. Here, demonstration will achieve more than words. At the U. S. Naval Hospital in Philadelphia we used to have a show for our amputees that included an exhibition of splendid ball-room dancing by a beautiful young lady and an older gentleman in evening dress. In the middle of the act the couple stopped a moment while the man rolled up both trouser-legs to show he was a double amputee with one above knee and one below-knee amputation. The effect on our patient was electric, immediate and lasting.

At this point let me emphasize a basic principle in rehabilitation: the goal of every handicapped person is to achieve as great a

degree as possible of **independence**, to do as much as possible for himself. But sometimes, because of the nature of the disability, this is impossible and he must accept some help. The question now becomes one of deciding whether to rely on another person and be **dependent**, or to attempt to use some type of mechanical aid or gadget which will permit him to be **independent**. **The aim of all rehabilitation should be independence.** If a gadget will bring that independence, the patient should have it called to his attention, and if possible, should be instructed in its use.

I shall now show you a book that is useful in helping the patient find the proper gadget: its title is **"Living with a Disability."** It was prepared by that outstanding exponent of rehabilitation, Dr. Howard A. Rusk and his associate, Eugene J. Taylor.[9] The pictures almost tell their own story.

Now, lest you think that all rehabilitation is a complicated thing at best, let me remind you that some of its most important aspects are the very simplest, and like so many simple things are so often neglected.

Obesity is the commonest disease in the United States. It is in itself the greatest single threat to longevity. Its relation to diabetes is of outstanding importance in those past middle years. Its prevention in the first instance and its effective control when present therefore offer the greatest reward in terms of long life and of being better able to bear most other handicaps that old age imposes. One cannot overstress the importance of girth control.

Osteoporosis can have many causes, endocrine and metabolic, but in advancing years a factor which can enter into every case is **disuse**: this may be due to pain of arthritis which leads to immobility, or simply the failure of an aging person to do things through lack of interest. Whatever other treatment may be called for, it will fail if it does not include such exercise of which the patient is regularly capable. I also hope that you wil not be guilty of increasing the osteoporosis, with eventual spontaneous fracture and complete invalidism by the **prolonged** administration of corticosteroids.

A program of high protein dieto-therapy will facilitate the anabolic processes so necessary in the rehabilitation of our aged patients, especially during periods of recovery from intercurrent illness or after surgical procedures.

The commonest prostheses that our aged need are **dentures**, **glasses** and **hearing aids**. The dentures and the glasses call for no special comment, but the hearing aids do. Most people are willing to acquire glasses and dentures when they need them, they go about it in a proper way, and they need no instruction in their use

But how many people who are more or less deaf, fail to get a hearing aid? When they finally do, why do they go to some layman whose only recommendation lay in the advertisement in print or by radio? And of course they receive very little instruction in how to use the gadget. To be sure, the time has not yet arrived when an otologist, after a complete audiometric examination can write a prescription for a hearing aid, to be filled in a place equipped to fill that prescription, as is now done for a pair of glasses. But we are approaching that stage: the otologist can advise, and the hearing aid "center" in many cities will supply the type of hearing aid best suited to the particular person. Then comes the important step of teaching the patient how to use the device under various circumstances and especially how best to use it to supplement what hearing he has left.

But the most important thing is again the most neglected: training in lip reading. Most people think of this as a highly specialized and difficult affair. That is by no means true. In the rehabilitation of our deaf service personnel, we found that adequate skill in lip reading could be acquired in 7 weeks. When some one objects by pointing out that these were young men and therefore more adept at learning, I can cite the case of our oldest patient, a naval captain in his upper fifties, who learned not only full face but profile lip reading, and within 5 weeks.

The aged are inclined to get careless about their feet. Therefore those who are responsible for these patients must give this matter special attention. A body is as strong as its feet, and so a **podiatrist** is a necessary member of a rehabilitation staff.

Now let me call your attention to another, and for most places a new aspect of rehabilitation of the aged: **the geriatric hospital.** It is not equipped with all the expensive facilities that a more acutely ill type of patient requires: complete surgical suites, diagnostic and therapeutic X-ray departments, laboratories to perform every conceivable test. These functions can be supplied when needed by transferring the patient to a general hospital, conveniently located on the same compound or across the street. It should have emphasis on physiotherapy, occupational therapy and rehabilitation. Its details of construction should be planned to meet the needs of the aged and handicapped: ramps, hand rails, easy stairways. There should be dining rooms for ambulatory patients and with opportunity for self-service. There should be out-patient departments that would serve as day hospitals and continued retraining centers for those who had been discharged to home to sleep.

There are two further important reasons why such geriatric

hospitals should be **separate** structures, rather than a ward or a wing of a general hospital. The obvious one is: the more efficient functioning of a staff completely dedicated to the problems of the aged. The other is an economic one: **separate cost-accounting** in an institution that will be decidedly less expensive to build and maintain than is the modern general hospital. When this point has been proved to a community, it will be easier to get other communities to build them.

In the meantime there are the possibilities of converting existing hospitals to such use. The advent of effective therapy for a number of conditions has shortened the hospital stay of such patients, so that beds for those conditions are or will be present in oversupply. The field of tuberculosis is a notable example. I am sure that with the conquest of poliomyelitis there will be needed fewer beds for crippled children.

You have exactly such a situation: At the time of my last visit you had just completed your fine new tuberculosis hospital. erected on the grounds of the old one, which in turn had been built when the death rate from tuberculosis in Puerto Rico was around 400 per 100,000. Today the death rate has dropped to less than one-tenth of that figure. As a result you are planning to exchange the **new** tuberculosis hospital with the District Hospital at Bayamón (which I saw dedicated in 1939). The **old** tuberculosis hospital will give way to a new medical center that will include the medical school.

I would suggest that now is the time to plan one building there as a pilot experiment for the rehabilitation of your aged. It might even be accomplished by the simple conversion of one of the old one-story structures now on the ground.

That this could succeed, I saw beautifully demonstrated in New Zealand last March. The 500 bed U. S. Army Hospital in Auckland and the 500-bed U. S. Naval Hospital near Wellington, both built during the last war, have been converted into geriatric hospitals that are doing an excellent job of rehabilitation. The success of these projects is due in large measure to the fact that admission to these geriatric hospitals is not directly from the community, but from the general hospitals of the area which attempt to select cases that give some promise of being capable of rehabilitation. I strongly recommend the idea for your consideration.

Then there is one more aspect of the problem: **the continuation of attention to the aged in their homes.** Everywhere there is happening the same trend in our living habits from 3-generation to a 2 generation families and smaller homes. More and more the elder people are living in their own quarters. Modern house-keep-

ing conveniences, easier transportation, better education, diversion by radio and television, pensions and social security income have combined to make our older citizens more desirous of, and better able to secure their independence. The longer they can maintain that independence, the happier they will be, and the less will be the burden on the taxpayer. Let me remind you: **there is no case on record where children in a court of law fought for the custody of a parent.**

Therefore the aged must be given such help as may be needed to remain independent. Last April in Melbourne, Australia, I saw an interesting application of this principle. Cooking becomes more and more of a chore for the aged, so that they easily fall into bad dietary habits. In Melbourne they have a "Meals on Wheels"[3] service, that delivers one hot meal a day to the homes of the aged who are house-bound, or find it difficult to eat out.

There are ever so many things that can be done in such community projects for the aged, not only for their comfort and pleasure, but for their gainful employment. I can envision programs maintained by churches, benevolent societies, labor groups, fraternal order, visiting nurse services and even Island Industries, Inc. This might offer employment for those displaced in industry by machines, as suggested by Sir George Thompson.[12] The important point is this: the good work begun in the rehabilitation service of your general hospitals and in your geriatric hospitals can be preserved and extended through such **extramural** programs.

For many years I have visited and revisited this beautiful island. I have observed with great interest the various problems that have confronted you in many fields: medical, social, economic and others. I have been impressed with the ingenuity, vision and steadfastness of purpose which you bring to the solution of those problems. Insularity presents serious problems of over-population, food supply and many things well known to you. It also has one important advantage: a rigidly controlled environment. There are fewer variables in your research problems, so you should arrive at sounder conclusions and earlier solutions.

I therefore look forward eagerly to future visits to see what you have accomplished in the rehabilitation of your aged, as well as in other fields, and to learn from your further experience.

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HYPOTHERMIA AND ITS COMPLICATIONS; METHODS OF CONTROL

R. W. RODRÍGUEZ, M.D., A. RIBERI, M.D., P. F. GRICE, M.D. and H. KAJIKURI, M.D.*

Since Bigelow[1] and Boerema[2] introduced hypothermia in experimental cardiac surgery, a great deal of work has been done in this field by many investigators. The main advantage ascribed to hypothermia, has been the reduction in oxygen requirements by the body tissues. It thus followed that a circulatory arrest would be of no harm to the brain, even if protracted for more than the three to seven minutes available under normothermic conditions. Experimental evidence accumulated in this direction, has shown that in moderately hypothermic dogs, the venous inflow occlusion could be extended up to fifteen minutes without any evidence of residual cerebral damage[1 2 4]. It was thus propounded that during the time of caval occlusion it would be possible to open the heart's chambers and attack interseptal defects and other lesions under direct vision. This has been shown to be feasible. There still remains, however, a great deal of uncertainty and difficulty due to what have been and still are considered the usual dangers and disadvantages of hypothermia, i.e. ventricular fibrillation, coronary and cerebral air embolism, limitation in the time of safe caval occlusion as it affects cerebral circulation, the indecision as to the role played by the extrinsic nerve supply to the heart. The ensuing discussion deals with the experience and results in more than four hundred experimental cardiac operations performed under hypothermia, in an attempt to develop and evaluate methods directed at counteracting the aforementioned complications.

I. VENTRICULAR FIBRILLATION

It was felt that before hypothermia could be safely applied to human beings, some method of reduction in myocardial irritability should be achieved. To do this, a technique was employed which in the hands of Lian and associates[5], had also proved successful in preventing ventricular fibrillation in dogs under drug induced hypothermia.

Two groups of dogs and controls were used in these experiments. In the first group, the animals were cooled to a rectal temperature of 30°C. by immersion in a bath of water and crushed

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From the Department of Surgery, Indiana University School of Medicine, Indianapolis, Indiana. Aided by grants from the Indiana Heart Foundation and the James Whitcomb Riley Memorial Association, and by a contract between the Office of Naval Research, the United States Navy and Indiana University.

* Present address: Department of Surgery, University of Puerto Rico School of Medicine, San Juan, Puerto Rico.

ice. This temperature usually dropped to a low of 26° to 28°C. by the time the thoracotomy was performed and the heart exposed. The chest was entered through the fourth intercostal space, the venae cavae were dissected free and the azygos vein ligated at its entrance into the superior vena cava. The pericardium was incised longitudinally, anterior to the phrenic nerve. During these maneuvers it was not unusual to observe extrasystoles and cardiac arrhythmias. The area of the superior vena cava — right atrial junction was injected with approximately 10-15°c. of 1% Procaine Hydrochloride. The effect on the heart rate, color and irritability were immediately noted. These changes consisted of slowing of the heart, improvement in the heart color, reduction of irritability to external manipulation and disappearance of the P wave or lowering of its voltage in the electrocardiographic tracing, suggesting evidence of sino auricular node blockade (Fig. I).

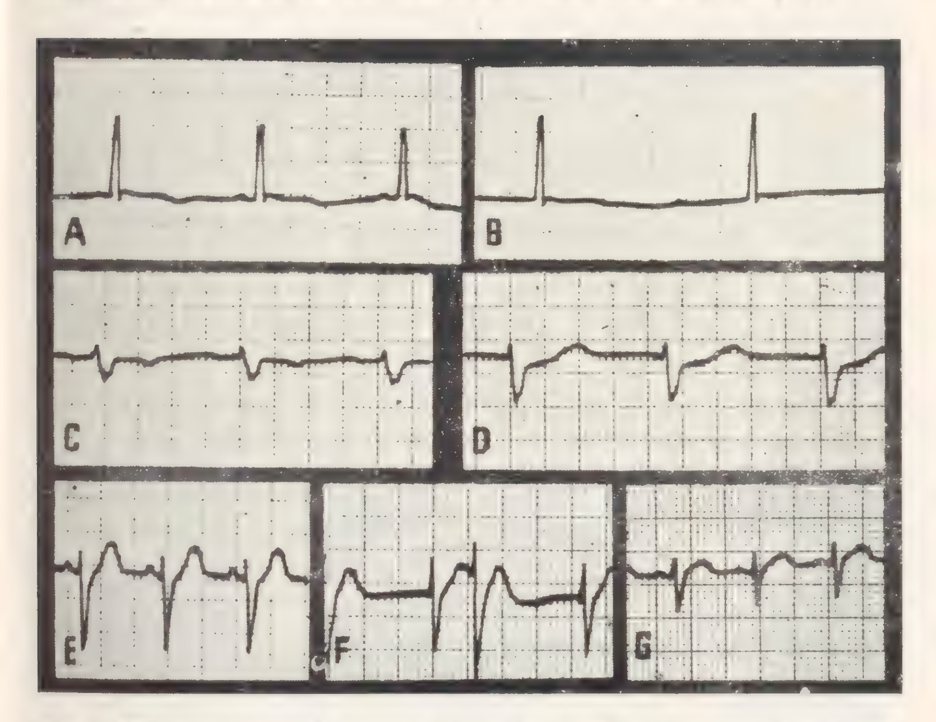


Fig. I: Electrocadiographic changes in Dog No. 28. Ventriculotomy and suture in interventricular septum.

A. At 27°C., before sino-auricular node injection.
B. Immediately after injection—absence of P wave, prolongation QRS interval.
C. Circulation resumed.
D. Chest closed.
E. At 35°C.
F. Two days post-operatively—Premature ventricular systoles.
G. Seven days later. No abnormalities in rhythm & conduction.

Following procaine blockade of the sino-auricular node, several stimuli were applied to the heart, including rough external manipulation of the heart, massage of the interventricular septum by a finger inserted in the right ventricle through the right atrium and tricuspid valve, and a wide ventriculotomy with the insertion of one or two silk sutures into the interventricular septum. The results of these stimuli were as anticipated in the control series. Ventricular fibrillation occurred in all (100%) of these animals. In contrast, none of the injected animals subjected to the same stimuli as well as ten to twelve minutes of caval occlusion, developed ventricuar fibrillation. In another group of dogs, the cooling was carried to 19º - 22.5ºC. and the same stimuli applied. In 56% of the controls in this group, ventricular fibrillation ensued. Of dogs protected by sino-auricular node blockade, 94% were free of ventricular fibrillation. This clear cut result has been corroborated by Radigan and co-workers.[7]

II. CORONARY AIR EMBOLISM AND VENTRICULAR FIBRILLATION

Attention was then directed to the protective qualities of procaine blockade of the right atrial wall in dogs in which coronary air embolism was being produced. It was found that procaine blockade delays somewhat the onset of fibrillation but cannot prevent it. The following method was then used in order to restore normal cardiac function.[5,9,12] It consisted of cross clamping of the ascending aorta, followed by strong cardiac massage for two to three minutes in order to force the air out of the coronaries. If air still remained entrapped, incision of the terminal coronary branches was performed, thus releasing the air. Stronger and coarser fibrillation was produced by intracardiac injection of one or two cc. of a 1:10,000 epinephrine solution, followed by further massage, and application of three successive electric shocks of 170 volts each and one tenth second duration.

This routine enabled the restoration of normal beat in all the animals. However, in the controls which had not received procaine blockade, fibrillation frequently recurred. In one such animal it recurred nine times and was finally stopped with blockade and electric shock. In none of the dogs with right atrial wall injection did fibrillation recur.

III. ROLE OF EXTRINSIC NERVE SUPPLY

In an attempt to better understand the reason for the value of procaine blockade and to gain further knowledge of the role played by the extrinsic nerve supply to the heart in ventricular fibrillation, several cardiac denervating procedures were carried out in ninety moderately hipothermic dogs subjected to ten minutes of venous

inflow occlusion, right ventriculotomy and passage of two sutures in the interventricular septum. (Table I)[5,10,12]

Table I — Denervating Procedures

Procedure	No. Animals	Fibrillation
Bilateral Upper Dorsal Sympathectomy and Stellate Ganglionectomy	15	0%
Bilateral Section of Cervical Vago-Sympathetic trunk	15	80%
Bilateral Division Vagus in Chest Below Caudate Ganglia	15	20%
Bilateral Upper Dorsal Sympathectomy, Stellate Ganglionectomy, Division of Cervical Vago-Sympathetic Trunk	15	40%
Intravenous Arfonad	15	13%
Electric Stimulation Vagus in Chest	15	17%

As can be noted from the observations in Table I, with the exception of the bilateral section of the vagi in the chest, sympathetic denervation or blockade efficiently protect the hypothermic animal from ventricular fibrillation.

With all this previous experience as a background, we were able to produce and repair ventricular septal defects in a series of hypothermic dogs without mortality, Figs. II & III.

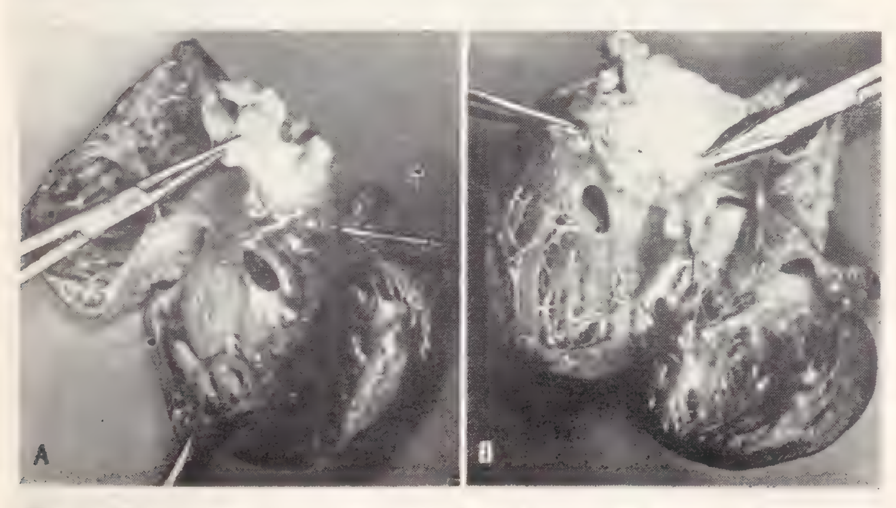


Fig. II: Experimental Inter-Ventricular Septal Defect, ten days after production.



Fig. III: Experimentally repaired Inter-Ventricular Septal Defect.
A. Seen from right side. B. Seen from left side.

IV. CORONARY AND CAROTID ARTERY PERFUSION

It was anticipated that the time allowed to work within the open heart would probably be too short to be applied to the more complicated defects of the human heart. Attention was then directed toward some technique able to give a prolongation of time for working within the heart chambers without producing irreversible brain damage. A method of perfusing the coronary and carotid arteries with oxygenated blood was devised.[12,13]

Dogs were cooled to 30°C. by the immersion cooling method, their temperature dropping to 26°C. by the completion of the thoracotomy. A polythene catheter was introduced into the left subclavian artery to the ascending aorta just above the coronary ostia. Following sino-auricular node blockade with ten to fifteen cc. of 1% Procaine Hydrochloride, complete caval occlusion was instituted. The perfusion with the oxygenated blood was begun and the animals were placed in Trendelenburg position to aid gravity perfusion of the brain. This perfusion given at the rate of 25-35 cc. per minute, has permitted the safe prolongation of inflow occlusion and the performance of a wide right ventriculotomy for times of from 29.5 to 39.5 minutes, with a very low incidence of ventricular fibrillation and with a very high survival rate.[13]

In thirteen of the forty one dogs used in this experiment, coronary perfusion alone was carried out, with all the dogs surviving periods of caval occlusion of from 25 to 29.5 minutes without apparent neurologic impairment. This seems to show that cerebral resistance to ischemia is much greater than supposed up to the present time and that myocardial oxygenation is much more important than anticipated.

We have recently used this technique of coronary and carotid artery perfusion in repairing atrial and ventricular septal defects in experimental animals.[14] Only two of twenty six animals operated upon to the present time and subjected to venous inflow occlusion for periods of from 29.5 to 35 minutes, have died. One death resulted from bleeding and one from brain damage following an overlooked cardiac arrest during repair of the chest wound.

CONCLUSION

Sino-auricular node blockade with Procaine, treatment of coronary air embolism and ventricular fibrillation, coronary and brain perfusion with oxygenated blood, have considerably increased in our hands, the over all safety of experimental open heart surgery under hypothermic conditions. These methods have efficiently added to the operative time allowed under hypothermic conditions.

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CYTOMEGALIC INCLUSION DISEASE

REPORT OF A CASE FROM PUERTO RICO

*VICTOR M. AREAN, M.D.**

Cytomegalic inclusion disease or disseminated salivary gland virus infection, is a fairly well delimited clinico-pathologic entity, the importance of which has become apparent only in the last two decades.

The disease affects chiefly premature children in the first year of life, the highest incidence being from birth to three months of age. It is characterized by jaundice, generalized petechiae or other hemorrhagic tendencies, a hemolytic type of anemia with large numbers of nucleated red cells in the peripheral blood, thrombocytopenia, splenomegaly and, more rarely, hepatomegaly. Pneumonitis is frequently, a part of the clinical picture. Death occurs as a result of respiratory complications, of hemorrhagic phenomena or, in isolated cases, because of renal insufficiency. In older children the picture, at onset, is that of a pneumonitis; in others the first clinical manifestation is jaundice with progressive derangement of liver function, complicated later on, by respiratory disease leading to death. In rare instances the child is stillborn or dies shortly after birth as a result of congenital malformations of the brain incompatible with life.

Pathologically the disease is characterized by the presence of gigantic cells, varying from 10 to 50 micra in diameter, containing intranuclear and intracytoplasmic inclusions. The intranuclear inclusion appears as a mass of reddish material separated from the nuclear membrane by a clear halo; this gives the cells a "bird-eye" or "owl eye" appearance. The nuclear chromatin is displaced towards the periphery and tends to accumuate into two or three nodules in the inner aspect of the nuclear membrane ("orbital bodies"). Because of the inclusion the nucleus is enlarged, so that it occupies half or more of the cell diameter. The cytoplasmic inclusions are multiple, tiny structures surrounded by a clear halo and usually conglomerated towards the free border of the cell. The sites most commonly affected by these changes are the epithelial cells of the excretory ducts of the salivary glands, the pancreas, small bile ducts, alveolar epithelium and the renal tubules. However they have been described in the heart muscle cells, in smooth muscle, fat cells, reticulo-endothelium, etc. In the central nervous system they are encountered mainly in astrocytes, but neurons and microglial cells may show identical alterations.

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* Associate Professor of Pathology, School of Medicine, San Juan, P. R.

At the present time the viral etiology of this condition is generally accepted. Cytomegalic inclusions have been found in a great number of animals, either as an incidental finding or while investigating the nature of some epizootics. The virus is highly species-specific so that transmission from one species to another has consistently proven negative. This fact besides greatly reducing the means for studying the biological characteristics of the organism, nullifies all attempts to use laboratory animals as a diagnostic method. Furthermore, the virulence of the virus is so low that although transmission from one animal to another of the same species has been accomplished, the attempts are successful up to the second or the third passage; afterwards the virulence of the causal organism is so exhausted that no ulterior positive results can be obtained. The virus has never been cultured in artificial media or in embryonated eggs. Smith and co-workers have recently reported success in culturing it by using special tissue culture technics.

It is beyond the scope of this paper to give a detailed account of the clinical picture or the etio-pathogenic and pathological aspects of this disease. For thoroughly satisfactory reviews the reader is referred to the papers of Smith and Velios, Wyatt and co-workers, Linzenmeier and those of Seifert and of Cappell and McFarlane.

We would like to emphasize that the disease is world wide in distribution and that a diagnosis can be made pre mortem. Because of the readiness with which the cytomegalic structures are shed, they can be identified in the urinary or spinal fluid sediment or in the sputum.

Although the clinical picture, chiefly in infants, strongly suggests that seen in erythroblastosis fetalis, the lack of immune antibodies in the child's serum and the frequent observation of Rh positivity in both the mother and the child, are enough to exclude the latter and to encourage a more thorough search along the lines of cytomegalic inclusion disease.

In the literature from Central and South America there are only two reports about this condition (Potenza), both from Venezuela. The recent observation of a case from Puerto Rico and a certain clinical and pathological peculiarities of it, has prompted us to inform the following case.

CASE REPORT

A four and one-half year old mulatto boy was admitted to the San Juan City Hospital on December, 19, 1952 because of jaundice, petechiae and fever of several days duration.

The child had been in good health until June of the same year, when he developed daily episodes of low grade fever, headache, anorexia and jaundice. Concomitantly, the mother noticed ecchymotic areas in the body and subconjunctival hemorrhages which she ascribed to trauma. At that time the patient was seen in the Out-Patient Department of the Hospital and was found to be jaundiced and to have an enlarged liver. He was given supportive therapy and given orders to keep in complete bed rest at home. A cephalin flocculation test was negative. On July 22, he was less jaundiced but the liver was still enlarged and an ecchymotic area was observed in one leg. The tourniquet test was positive; bleeding, clotting and clot retraction times were normal. On August 12, the child was examined again at which time the jaundice and ecchymosis had disappeared and the child was feeling well. During the last week of November, however, he developed malaise, anorexia and jaundice. He was hospitalized at another hospital for that reason but he developed a generalized rash and was discharged because the physicians thought he had measles. He continued to be icteric and the rash persisted. Then he developed a cough and experienced a daily rise in temperature. There was no vomiting, diarrhea or bleeding. The urine and feces were of dark yellow color. Past history revealed he had had measles at one year of age and had suffered from frequent sore throats. Physical examination showed a temperature of 103º, pulse 122 and respirations 22 per minute. The patient was a well developed thin, mulatto boy who appeared acutely ill and intensely dyspneic. There was a generalized, discrete, purpuric rash throughout the body. The pupils and fundi were normal. The conjunctiva were markedly injected and the sclerae deeply jaundiced. There was flaring of the alae nasi on breathing. The lips and tongue were dry and the pharynx mildly injected. Small lymph nodes were palpable in the neck. The lungs were resonant and the breath sounds were rough. There was tachycardia. The liver was palpable 2 fingerbreadths below the xiphoid process and was non-tender. The spleen was not felt. The rest of the examination was normal. Laboratory findings were as follows: Redcell count 3.700.000, hemoglobin 68 per cent., white cell count 11.200; the differential count showed 80 per cent segmented, 17 lymphocytes and 3 eosinophils. On December 24 he developed subcutaneous emphysema and rales in both lungs with spiking temperatures ranging from 100º to 103º. An x-ray of the chest showed moderate degree of subcutaneous emphysema in the left axilla and left upper chest; the lungs appeared normal. Three days later the emphysema had subsided and the jaundice began to clear. However the liver was still palpable. On December 29 he developed again an increase in tem-

perature up to 104º, this persisted until death. The patient became very pale, bled easily from veni-punctures and petechiae appeared in the skin, conjunctiva, buccal mucosa and the pharynx. On the following day blood counts were repeated and revealed red blood cells 1.450.000, hemoglobin 22 per cent, white cells 10.650; the differential was lymphocytes 76 per cent, eosinophils 24 per cent; there were 14 normoblasts per 100 white cells. There was alsso anisocytosis, and slight microcytosis. On January 12, 1953 the red cell count was 560.000, hemoglobin 10 per cent, white cell count 6,050 and the reticulocyte count 3 per cent. A platelet count revealed 54.000. The clotting time was 3 minutes and 30 seconds and the bleeding time 2 minutes and 17 seconds. The fragility test was reported as follows: Control: hemolysis began at 0.40 per cent and was completed at 0.28 per cent; patients blood: hemolysis began at 0.44 and was completed at 0.30 per cent. The Coomb's test was negative. A bone marrow study revealed 4 per cent neutrophilic myelocytes, 10 per cent neutrophilic metamyelocytes, 14 per cent neutrophilic stabs, 27 per cent neutrophilic segmented forms, 21 per cent eosinophils, 1 per cent pronormoblasts, 0.5 per cent basophilic normoblasts, 0.5 per cent megakaryocytes and 22 per cent lymphocytes. The smears were described as showing normal activity. Prothrombin time control 14.3; patient's 16.4 seconds. The total cholesterol was 175 mg. per 100 ml.; thymol turbidity 0.7 units cephalin flocculation 2+ in 48 hours. Bilirubin 0.72 in one minute and 1.9 mg. in 30 minutes. Urinalysis was negative. Repeated blood cultures were negative. Agglutination for typhoid, paratyphoid and brucella were also negative. The patient was treated from the time of admission with blood transfusions, vitamin K and antibiotics as well as parenteral fluids. Despite all measures his condition deteriorated progressively; he became apathetic, unresponsive and expired on January 12, 1953, seven months after the onset of symptoms. At autopsy the body was that of a well developed poorly nourished mulatto boy showing generalized petechiae and subconjunctival hemorrhages. There was an ecchymotic area over the posterior third of the tongue measuring 3 cm. in diameter. Similar petechiae were found in the pleura, peritoneum, pericardium and over the visceral pleura of the lungs, the epicardium, myocardium and endocardium. The lungs were moderately congested and their cut surface revealed scattered foci of hemorrhage varying from 1 to 4 cm. in diameter. A bloody froth exuded from the cut surface. The spleen and pancreas appeared grossly negative. The liver weighed 510 gm. and was seen protruding for a distance of 4 cm below the right costal margin. Its surface was normal, but the cut surface showed blurred structural markings and had a yellow-gray color. The kid-

neys, lower urinary tract genitalia, adrenals and brain were negative. Histologic examination of the heart revealed scattered foci of fresh hemorrhage chiefly in the epicardial region extending to the muscle fibers. In the lungs there was a diffuse infiltration of the interstitium by lymphocytes, monocytes and histiocytes with broadening of the alveolar septa. The alveoli contained variable amounts of macrophages, mononuclear cells and fibrin. The most striking finding was that of large cells present either in the lumen of the alveoli or attached to the septa. They varied from 15 to 30 micra. The nuclei was large and contained a round or ovoid, reddish inclusion surrounded by a clear halo. The nuclear chromatin was displaced towards the periphery and grouped into two nodules in the inner aspect of the nuclear membrane. There were occasional binucleated cells in which case both nuclei contained inclusions. The cytoplasm was foamy and abundant; in some cells there were tiny intracytoplasmic structures surrounded by a clear halo and disposed towards the free edge of cells (Fig. 1). It was possible to identify cells of normal size in which the nucleus contained a minute inclusion; otherwise these cells appeared unaffected by the inclusion. However transitions from these to the

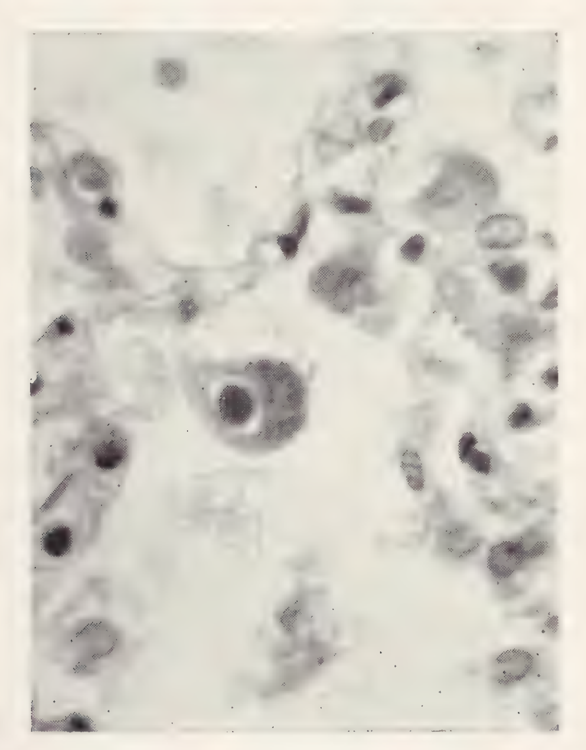


Fig. 1.—Lung. Cytomegalic structure showing intranuclear and intracytoplasmic inclusion. PTAH stain, 800 X (approx.)

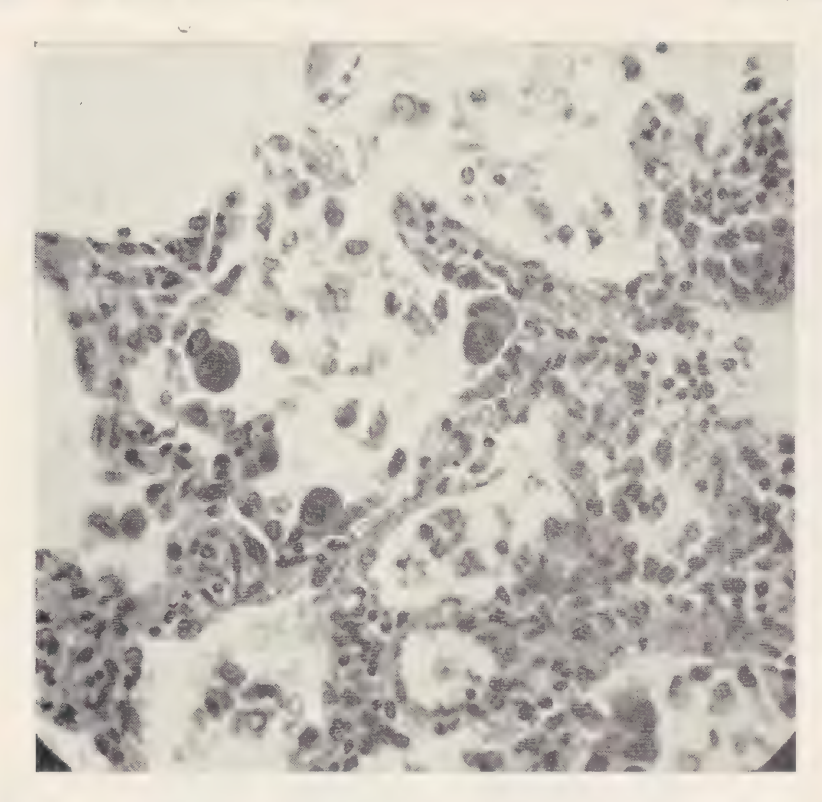


Fig. 2. Lung. Observe cytomegalic structures free in the alveolar lumen or attached to the septal wall. Hematoxylin-eosin, 100 X.

larger ones were readily encountered (Fig. 2). Many of the cytomegalic structures were desquamated in the alveolar spaces; some were found mixed with mucous material in the lumen of larger bronchi. The spleen showed hyperplasia of the reticulo-endothelial elements, foci of hematopoiesis and occasional cytomegalic structures. The pancreas showed groups of cytomegalic elements without any inflammatory reaction about them. In the liver there was a pronounced broadening, fibrosis and infiltration by round cells of the portal space. As a result there was marked distortion of the liver architecture, suggesting in some areas, an early stage of cirrhosis. The epithelium of some bile ducts contained intranuclear inclusions. Similar findings could be detected in isolated hepatocytes, mainly at the periphery of the liver lobules, (Figs. 3 and 4). In the gastroenteric tract the only finding of interest was that of a cytomegalic structure in the membrana propria of the stomach, without any inflammation in the surrounding tissue. The adrenals, kidneys, lower urinary tract, genitalia and the brain and pituitary gland were negative. Cytomegaly was found in the submaxillary and parotid glands, the thymus, lungs, spleen, pancreas, liver and

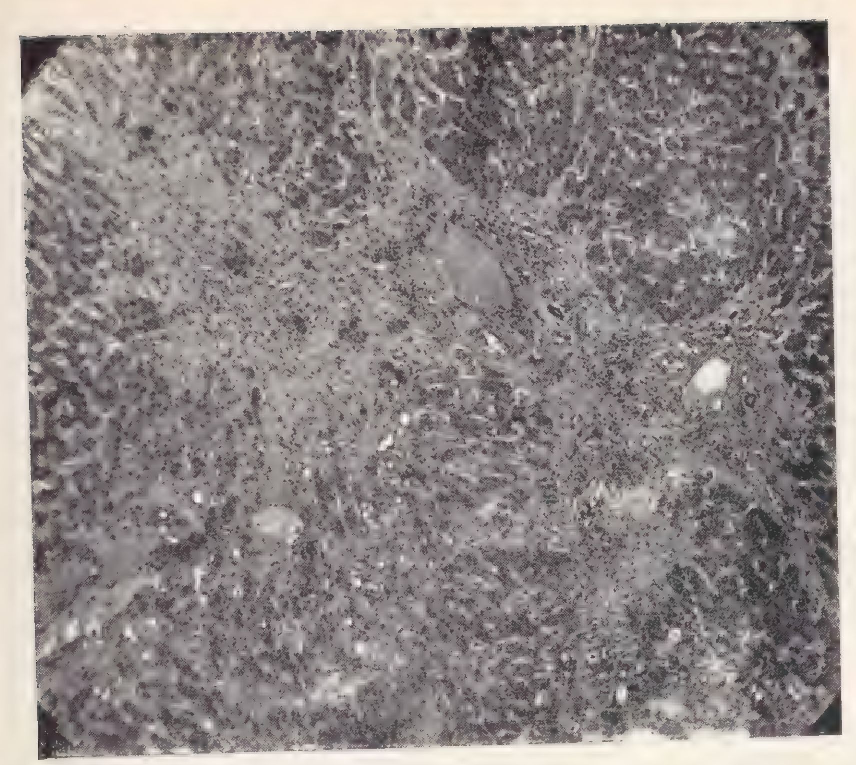


Fig. 3. Liver. Pronounced periportal fibrosis and round cell infiltration secondary to cytomegalic virus. Hematoxylin-eosin 100 X.

Fig. 4. Liver. Cytomegalic structure in a bile duct. Note the marked fibrosis of portal space.

mesenteric lymph nodes. The latter also showed foci of hematopoiesis. The bone marrow showed severe hypoplasia of all series, with relative increase in the number of eosinophilic granulocytes. Megakaryocytes were not identified despite multiple sections taken from the sternum, lumbar vertebrae and femur. There were some cells with abundant acidophilic cytoplasm and pycknotic nuclei barely suggesting elements from the megakaryocytic series; however, neither their size nor their structural characteristics were enough to warrant classifying them as such. Examination of other organs where foci of hematopoiesis was seen, failed to show megakaryocytes. The bone marrow was focally replaced by young fibroblasts and occasionally by masses of fibrin and or hemorrhage. Cytomegaly was not encountered in the marrow sections.

COMMENT

This case is of interest from several points of view, clinical and pathological.

In most of the cases reported the disease affected children less than three years of age. To our knowledge ours is the first instance in which the disease has had its onset at the age of 4 and one-half. Moreover, a review of the literature reveals that the course of the disease was a rapid unremitting one, the average survival from onset of symptoms being about three months. In our case the clinical course was characterized by several episodes of jaundice and or hemorrhagic tendencies, remitting spontaneously for short periods of time, and culminating in a picture of profound blood dyscrasia and respiratory manifestations six months after the first symptoms were observed. It is worthy of note to mention that in cases of cytomegalic inclusion disease reported in adults, there has always been an associated disease, usually of neoplastic origin, preceding the onset of the cytomegalic dissemination. For these cases it is postulated that the chronic, neoplastic process created favorable circumstances for the dissemination of a dormant salivary gland virus. The autopsy of our case failed to reveal any evidence of neoplastic disease or lesions that could not be related to this viral entity.

There are about seven cases reported in the world literature in whom the clinical manifestations were those of progressive derangement of the liver function; necropsy of these cases showed severe fibrosis, classified in some as true cirrhosis of the liver. The patient herewith reported developed jaundice from the onset, yet at no time were the liver function tests abnormal. This indicated that the jaundice was more of a retention type, than secondary to liver cell damage; histologic data corroborated this impression.

The immediate cause of death must be ascribed to the severe damage caused by the virus upon the bone marrow and in part to the pulmonary affection. Over a period of less than one month the blood count dropped from nearly normal values and normal bone marrow activity to red cell counts of less than one million and a platelet count of 54,000. At autopsy it was apparent that although all series had been affected, the megakaryocytes suffered more, for no such elements were found in multiple sections. Marked thrombocytopenia is not an unusual finding in many infectious and toxic processes. Less common is the observation of amegakaryocytosis. The damage to the megakaryocytic series is generally limited to an interference with the maturation of the megakaryocytes resulting in a decrease of blood platelets. But even in those cases it is always possible to identify megakaryocytes producing platelets, although they may be immature cells such as promegakaryocytes (de la Fuente[2]). In our case, as mentioned above no such cells were recognized.

The case is of interest also in that despite the severity of the affection of the lungs, liver, salivary glands, etc., the kidneys (one of the organs most commonly affected) was free of lesions.

It is possible that the diagnosis of the disease could have been made during the patient's life. Cytomegalic inclusion elements were identified in the mucous secretions of the larger bronchi at post-mortem, so that one would expect to find them in the sputum, had they been looked for.

Our case is the first one recorded from Puerto Rico indicating that more will be found in the future.

SUMMARY

A case of disseminated salivary gland virus infection in a Puerto Rican boy of 4 and one-half years of age is reported. The clinical picture was one of jaundice, hemorrhagic phenomena and progressive anemia and thrombocytopenia complicated finally with respiratory disease. Autopsy findings revealed marked fibrosis of liver, interstitial pneumonitis and hypoplasia of bone marrow with amegakaryocytosis. Cytomegalic structures were identified in the submaxillary and parotid glands, the thymus, lungs, pancreas, spleen, liver, membrana propria of stomach and in the mesenteric lymph nodes. Focci of extramedullary hematopoiesis were detected in the spleen and mesenteric lymph nodes. The kidneys were normal.

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SOME EFFECTS OF HYPOTHERMIA ON HYPOVOLEMIC SHOCK IN ANESTHETIZED DOGS

*GUMERSINDO BLANCO, M.D. and AGUSTIN FERNÁNDEZ, B.S.**

A study of the effects of hypothermia on hypovolemia in anesthetized dogs was initiated at the Laboratory for Experimental Surgery at the University of Puerto Rico Medical School in December 1954. A preliminary report on this study dealt with our early experiences in the field and the evolution of an experimental technique employing a modification of Walcott's method[1] of producing hypovolemic shock as the standard procedure upon which the effects of hypothermia were to be observed.

METHOD

Adult mongrel dogs ranging in body weight from 6.8 to 15.6 kg. were selected and their hematocrits, plasma volumes, and circulating blood volumes determined (by the T-1824 method). They were anesthetized using intravenous Nembutal (30 mgm. per kg. of body weight) and heparinized. The animals were secured in the supine position and both femoral arteries, and a carotid artery were exposed and catheterized using No. 21 plastic oxygen catheters. At this point three different procedures were followed.

(1) In twelve normothermic controls the animals were bled into sterile flasks containing heparin, at such a rate that dripping would occur between five and ten minutes after the onset of the hemorrhage. At this time spontaneous respirations would cease and the dogs would be intubated and attached to a mechanical respirator (Pneophore) for the duration of the experiment. Bleeding would proceed very slowly from this point on, and a continuous electrocardiogram during the remaining period of hemorrhage would show a tachycardia to be present usually changing into a bradycardia which would terminate in standstill or fibrillation if bleeding was continued. To prevent the death of the animal, 25% of the volume of blood extracted would be replaced rapidly by injection into the exposed carotid artery. Bradycardia would cease and if standstill had occurred tracings would reappear, showing a return to a faster pulse which would persist during the period of reinfusion. After 25% replacement was complete and spontaneous respirations were established the animals were observed for a period of six hours and, if alive at that time, were returned to their cages. EKG

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* From the Department of Surgery and the Laboratory for Experimental Surgery, University of Puerto Rico Medical School.
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tracings were taken during this observation period if any untoward signs became evident. In six of the controls intubation was carried out for periods ranging between 30 and 60 minutes before hemorrhage was started in order to determine the effects of early intubation and artificial respiration necessary in hipothermic dogs as will be described below.

In fourteen dogs, the same bleeding procedure was carried out with intubation and artificial respiration at the time of respiratory failure due to hemorrhage. After 25% replacement, these animals were cooled to temperatures ranging between 24º-26ºC using a refrigeration unit with blankets or immersion in water at 0º-5ºC. Hypothermia at the levels described was maintained for an additional hour at which time the animals were rewarmed and observed as were the controls. During the cooling and rewarming periods EKG tracings were obtained every fifteen minutes and whenever abnormal muscular or respiratory movements were observed.

Seventeen dogs were cooled to 24º-26ºC prior to hemorrhage and then subjected to the shock procedure. It was noticed in earlier experiments that intubation and artificial respiration had to be carried out as the temperature levels went below 28º-29ºC inasmuch as respirations were inadequate or absent at these temperatures. All of these animals therefore were intubated and attached to the mechanical respirator (Pneophore) for periods ranging 30 to 60 minutes before bleeding was started. Again, after 25% replacement, these dogs were observed for one hour during which time their temperatures ranged between 24º-26C, and at the end of this period they were rewarmed, observed, and later the survivors returned to their cages. In six of these hypothermic dogs, 25% replacement was not carried out for reasons explained below.

RESULTS

A summary of the data regarding weights, hematocrits, plasma volumes, control circulating blood volumes, maximal bleeding volumes, final bleeding volumes, and lengths of survival, is presented in the accompanying tables.

In general it was found that dogs rendered hipothermic before the bleeding episode could not be bled as rapidly as the normothermic controls, and in the cold animals the bleeding took 10 to 15 minutes to be completed, as compared with 5 to 10 minutes in control animals.

Electrocardiographic changes in hypothermic animals subjected to hemorrhage were similar to those shown by control animals except for the initial bradycardia due to the hipothermia per se.

As we have described above we relied on electrocardiographic demonstration of profound bradycardia to stop the bleeding and start 25% transfusion. As hipothermic dogs had usually bled less than normothermic dogs at this point, it was felt that perhaps maximal bleeding volumes had not been obtained as yet and for this reason, six hipothermic dogs were subjected to hemorrhage up to the appearance of extreme bradycardia at which time bleeding was stopped but no replacement was given. Three dogs died immediately from ventricular fibrillation, the other three died at 30 minutes, one hour and two hours respectively.

A comparison of average maximal bleeding volumes (expressed in percentages of control circulating blood volumes) between normothermic and hipothermic dogs shows the values for the latter to be 12% less than those obtained in the former. The same difference is observed when bleeding volumes per kilogram of body weight are compared in these two groups (normothermic 45.7 cc kg., hipothermic 33.7cc kg.) These findings seem to parallel those of Rodbard[2], and D'Amato[3], who have described substantial (up to 30%) reductions in circulating plasma volumes in hypothermic animals. Their observations were carried out at temperatures in the range of those employed in our experiments.

The mortality of normothermic controls was 80% within 24 hours, the average duration of survival after hemorrhage and 25% replacement being 7 hours. Hypothermia induced before or after the bleeding did not seem to affect these figures favorably, on the contrary the 24 hour mortality appeared slightly increased. (85% when animals were cooled after hemorrhage, 90% when hypothermia preceded the bleeding).

DISCUSSION

A number of observers have reported beneficial effects of hypothermia on hipovolemic shock as produced by various experimental procedures. These effects have been described as an increased tolerance to the bleeding episode and a reduced mortality rate when compared to those of normothermic controls.

The investigations presented herein fail to substantiate these findings, however differences reported may very well be due to variations in the experimental techniques employed.

As far as we know these are the first observations reported on the effect of hipothermia on dogs subjected to the Walcott procedure of producing hemorrhagic shock.

Again, the temperature levels attained in our hipothermic dogs (24º-26ºC) are lower than those employed by most workers in this

field. It is quite possible that varying degrees of hipothermia vary significantly in their qualitative effects on the shocked animal, and that the beneficial effects of minor (4 to 6 degrees) reductions in temperature may disappear or actually become deleterious when moderate (10 to 12 degrees) or marked (12 to 20 degrees) reductions in body temperature are employed.

A number of extraneous factors reportedly affecting the response to hemorrhage by themselves are introduced by these last reductions in temperature. In animals anesthetized with nembutal, intubation and artificial respiration become necessary below 28°C. Artificial respiration particularly when associated with positive pressure has been described as affecting the shocked animals unfavorably[7], and as a matter of fact was used as a standardized shocking procedure by Henderson[8] some years ago. For this reason some of our control animals were subjected to intubation and artificial respiration with the mechanical respirator for periods comparable to those used in hipothermic dogs. No deleterious influence of this particular procedure could be detected.

Perhaps the most significant concept to be derived from this study is that more extensive investigations will have to be carried out before the proper role of the hipothermic effect on hipovolemia is clear and especially before any practical application of cooling can be advocated on a sound experimental basis. In this last respect we have found the Walcott procedure particularly suitable, as the shock producing insult can be produced in a short period of time, and hipothermia may then be induced very much as it would have to be applied in clinical practice.

CONCLUSIONS

A study has been carried out on some effects of hipothermia on a standardized hemorrhagic shock procedure, (modified after Walcott's) in anesthetized dogs.

Hypothermia has been induced both prior to, and immediately following the shock procedure and the results in these two series of animals have been compared to those obtained in normothermic controls.

Moderate reductions in the maximal bleeding volume of hypothermic dogs have been observed. These findings are discussed.

No beneficial effect could be demonstrated in the use of hypothermia in the range employed (24° to 26°C) under the conditions of these experiments.

Further studies are absolutely necessary before the effects of hypothermia (and particularly varying degrees of it), on hypovolemic shock can be appraised correctly.

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(Tables in following pages)

TABLE I

HEMORRHAG E—CONTROLS

DOG No.	Wt. Kg.	Hct	P. V.	Control Circul. Blood Vol.	Maximal Bleeding Volume	M. Bl. V. C. C. B. V. %	M. Bl. V. Wt. Kg.	Returned	Final Bleeding Volume	F. Bl. V. C. C. B. V. %	F. Bl. V. Wt. Kg.	Survival Time post Hemorrhage
C.S.1	8.5	39	451	720	457	63	53.7	114	343	48	40.3	1 hr.
C.S.2	9.6	47	685	1248	460	37	47.9	125	335	27	34.8	10 hrs.
C.S.3	10.8	51	535	1048	620	59	57.4	150	470	45	43.5	7 hrs.
C.S.4	?	48	544	1008	600	60	?	150	450	45	?	24 hrs.
C.S.5	11.1	48	630	1168	550	47	49.5	135	415	36	37.4	8 hrs.
C.S.6	10.0	39	446	713	500	70	50.0	125	375	53	37.5	48 hrs.
C.S.7*	12.3	42	438	734	395	54	32.1	90	305	42	24.7	1/2 hr.
C.S.8*	11.6	47	403	734	410	54	35.3	100	310	42	26.7	1/2 hr.
C.S.9*	8.2	45	283	498	300	60	36.5	75	225	45	27.4	48 hrs.
C.S.10*	10.0	37	392	608	475	78	47.5	120	355	58	35.5	4 hrs.
C.S.11*	10.2	46	416	745	400	54	39.2	100	300	40	29.6	8 hrs.
C.S.12*	12.2	45	479	843	400	47	32.8	100	300	35	24.6	48 hrs.
Average						57	43.8			43	32.9	

* - Intubated and attached to respirator for 1/2 - 1 hr. before hemorrhage.

TABLE II

HEMORRHAGE PLUS HYPOTHERMIA

DOG No.	Wt. Kg.	HCT.	P. V.	Control Circul. Blood Vol.	Maximal Bleeding Volume	M. Bl. V. / C. C. B. V. %	M. Bl. V. / Wt. Kg.	Returned	Final Bleeding Volume	C. C. B. V. / F. Bl. V.	F. Bl. V. / Wt. Kg. %	Survival Time Post Hemorrhage
H.S.1	10.4	43	808	1376	765	55	73.5	150	615	44	60.6	End of Bleeding
H.S.2	9.0	54	472	980	430	45	47.7	100	330	34	36.6	1/2 hr.
H.S.3	9.0	40	451	732	450	61	39.9	120	330	45	36.6	22 hrs.
H.S.4	9.0	37	781	1211	530	44	58.8	130	400	33	44.4	12 hrs.
H.S.5	10.9	37	520	806	400	50	36.7	100	300	37	27.5	1/2 hr.
H.S.6	9.0	38	614	966	480	50	53.3	120	360	37	40.0	2 hrs.
H.S.7	8.0	48	439	814	450	55	56.5	112	338	41	42.2	12 hrs.
H.S.8	?	37	536	831	475	57	?	118	357	43	?	48 hr .
H.S.9	?	44	536	928	440	47	?	110	330	35	?	20 hrs.
H.S.10	9.2	41	550	907	480	52	52.1	120	360	40	39.1	12 hrs.
H.S.11	12.2	53	538	1095	550	50	45.0	135	415	38	34.0	12 hrs.
H.S.12	10.0	36	507	774	360	47	36.0	90	270	35	27.0	3 hrs.
H.S.13	9.3	45	439	773	390	50	41.9	98	292	38	31.4	3 hrs.
H.S.14	10.0	34	580	861	490	56	49.0	122	368	43	36.8	48 hr .
Average						51	47.5			39	38.0	

TABLE III

HYPOTHERMIA PLUS HEMORRHAGE

DOG No.	Wt. Kg.	HCT.	P. V.	Control Circul. Blood Vol.	Maximal Bleeding Volume	M. Bl. V. C. C. B. V. %	M. Bl. V. Wt. Kg.	Returned	Final Bleeding Volume	F. Bl. V C. C. B. V. %	F. Bl. V. Wt. Kg.	Survival Time Post Hemorrhage
H-1	11.0	50	643	1236	300	24	27.2	75	225	18	22.7	20 hrs.
H-2	6.8	48	369	684	265	39	38.9	65	200	29	29.0	End of Bleeding
H-3	10.8	43	641	933	400	44	37.0	100	300	32	27.8	2 hrs.
H-4	10.7	45	572	1008	370	37	34.5	95	275	27	25.7	20 hrs.
H-5	10.1	39	617	987	280	28	27.7	70	210	21	20.8	34 hrs.
H-6	10.0	43	404	683	390	56	39.0	95	295	43	29.5	10 hrs.
H-7	9.5	40	383	622	300	48	31.5	75	225	36	23.6	End of Bleeding
H-8	10.2	35	476	761	350	46	34.3	90	260	34	25.4	1/2 hr.
H-9	13.3	48	560	1039	550	53	41.3	135	415	40	31.2	1 hr.
H-10	8.5	34	480	726	525	72	61.7	130	395	54	46.4	6 hrs.
H-11	10.3	51	322	631	275	44	26.7	65	210	33	20.3	1/2 hr.
H-12*	15.6	44	763	1220	460	38	29.4	0				2 hrs.
H-13*	9.0	50	455	875	275	31	30.5	0				End of Bleeding
H-14*	8.0	49	392	740	325	44	40.6	0				"
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1. Overall, J. C.: Southern M. J. 47:789, 1954. 2. Editorial: New England J. M. 246:111, 1952.
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DE LA

ASOCIACION MEDICA DE PUERTO RICO

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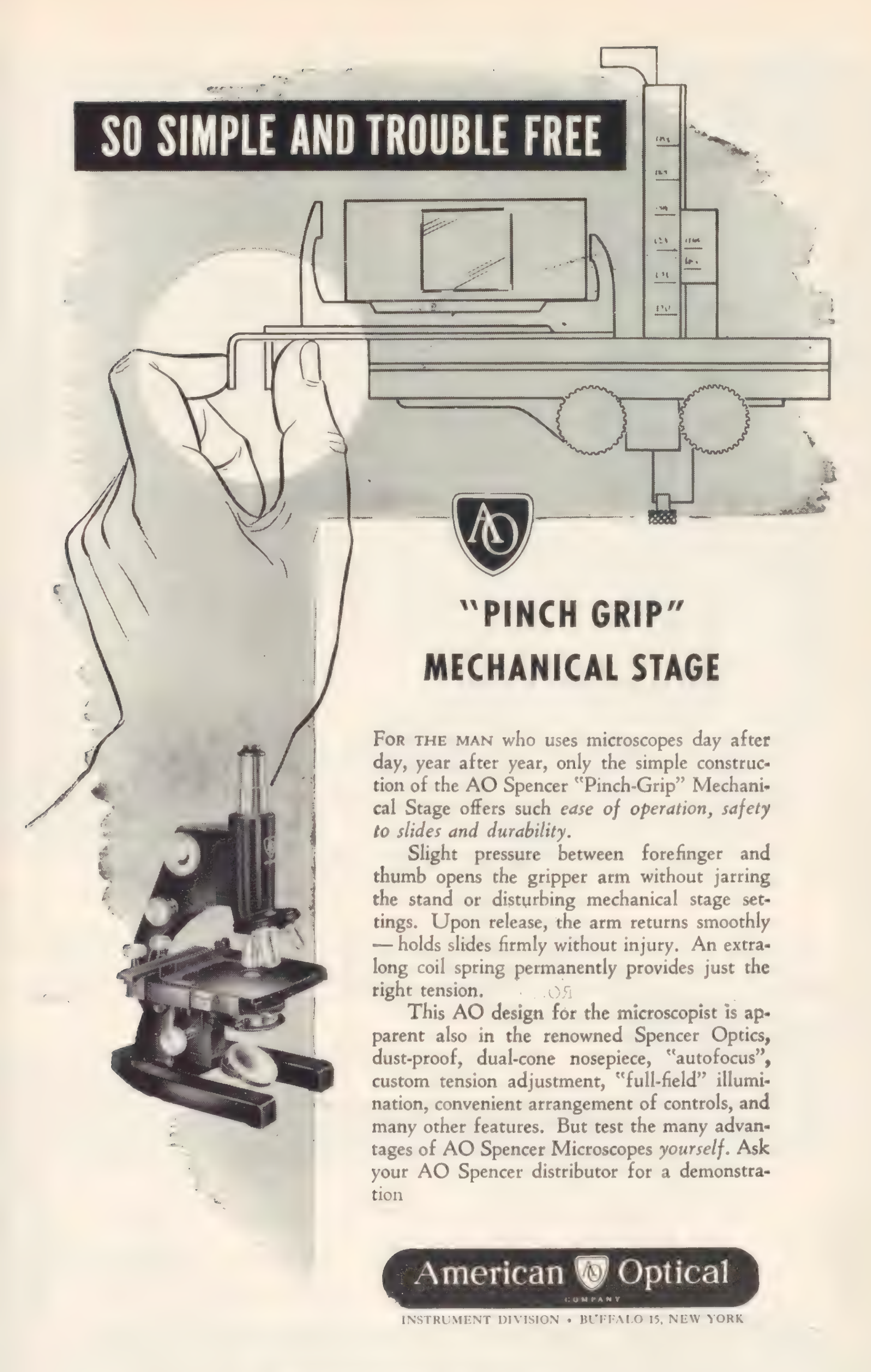
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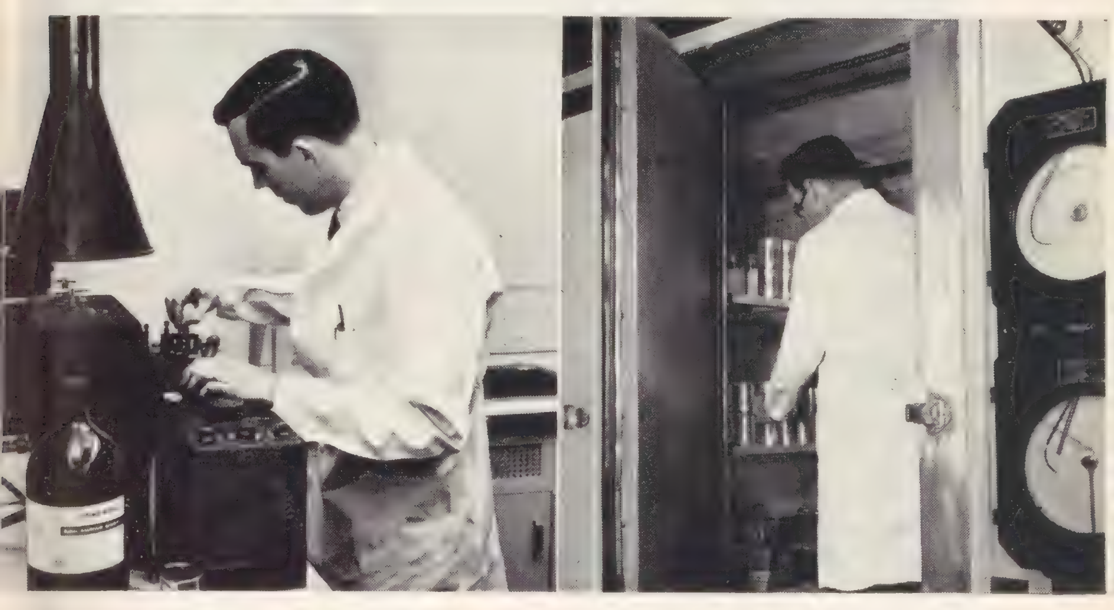
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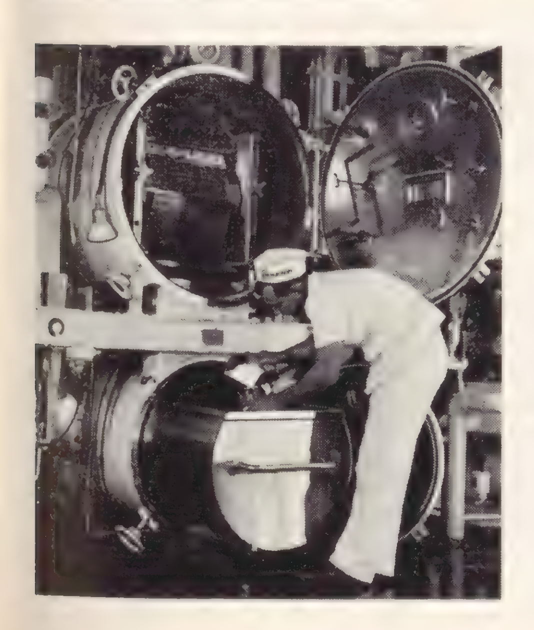
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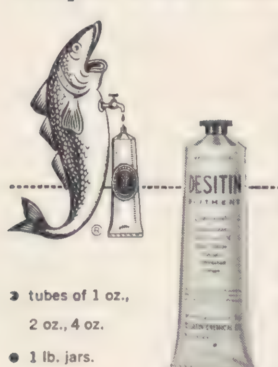


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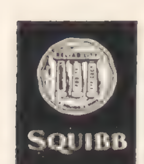
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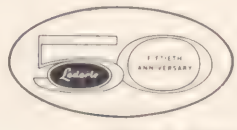
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DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 48 AGOSTO, 1956 NO. 8

THE RESPONSIBILITY OF THE RADIOLOGIST IN CHEST DISEASES

*LASZLO EHRLICH, M.D.**

Every clinician is cognizant of the fact that in a large number of chest diseases the patient is symptomless and the diagnosis is made in these cases entirely on the basis of the routine chest film. Even when there are symptoms such as fever, cough or hemoptysis, the clinician sometimes cannot decide whether the patient suffers from Tb., Pneumonia or Ca., just to mention the most important diseases. Again, the chest film is supposed to give the answer.

When the x-ray findings are more or less obvious, there is no problem. Not infrequently, however, one deals with a so-called "borderline" case in which the x-ray findings are so minimal that one is inclined to give a "negative" report. One could call this "underreading". Now, the same film viewed by another radiologist (or by the same radiologist on another day. . . .) may be reported as having an abnormality of serious import. In this case, one might use the term of "overreading".

When an obvious change develops in the due course of time in these so-called "borderline" cases, the radiologist reviews the old films, sometimes with embarrassment.

Of course, from the patient's point of view, the responsibility of making the correct x-ray diagnosis assumes a formidable magnitude. Did I say x-ray diagnosis? Well, I wish to emphasize that the term diagnosis should never be used by the radiologist. The x-ray finding is not a diagnosis. It is a diagnostic impression. Its sole purpose is to transmit information to the clinician who on the basis of this information can initiate the necessary clinical investigation. The final clinical diagnosis will be established by correlating the clinical history, physical examination, laboratory tests and x-ray reports; the relative importance of each of these will

* Chief, X-ray Service, San Patricio V.A. Hospital.
Presented at the Annual Meeting of the Puerto Rico Medical Association, December 10, 1955.

vary according to the case. Even if the information submitted by the X-Ray Dept. happens to be of decisive importance by disclosing an unsuspected lesion, it still remains a diagnostic impression and not a diagnosis.

In this hospital, I don't think I have ever used the term "diagnosis" in an x-ray report; I do use it in a loose fashion in the course of a conversation.

To make my point clear I want to give you an example. A few months ago we had a patient whose routine chest film showed the textbook picture of Beck's sarcoid. All the clinical investigations were negative. The clinician in charge wanted to discharge the patient with the diagnosis of sarcoid. I disagreed. Upon my insistence, he was discharged with the final diagnosis of "Bilateral hilar adenopathy, of c. u.".

In the following presentations, I would like to demonstrate to you the inherent difficulties of interpretation in making a definite diagnostic impression and also demonstrating that the responsibility of the radiologist begins and ends by the correct interpretation of the x-ray findings. The full responsibility of the final diagnosis, however, rests with the clinician.

Case #1

M. E. Age: 23

This patient was admitted to a hospital in 1946 for itchy swelling of his right little finger. History of pneumonia in 1945 followed by frequent bloody expectorations. The chest x-ray was reported as "showing thickening of the right hilar shadow, indicating a hilar type of Tb". 3 sputa were negative for Tb. On the basis of the x-ray report the diagnosis of "Chr. pulm. Tb., active" and he was transferred to a Tb. sanatorium.

Between 1946 and 1955, this man had spent 1374 days in different hospitals (23 admissions). 71 sputum examinations including cultures were negative for Tb. One single positive smear was reported by one small clinic in 1947. The same year he had artificial pneumothorax and phrenic crush.

In 1949 he was admitted to San Patricio Hospital where a bronchogram demonstrated slight bronchiectasis in his R. U. L. Since, he developed a severe conversion reaction, hypertension, glomerulonephritis and Laennec's cirrhosis. The latter was probably on alcoholic basis.

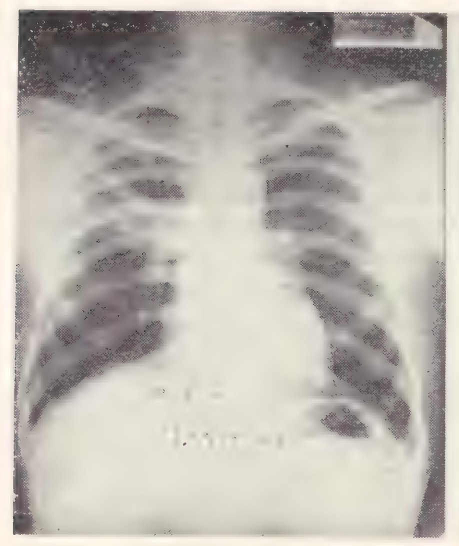


Fig. 1. — Case #1

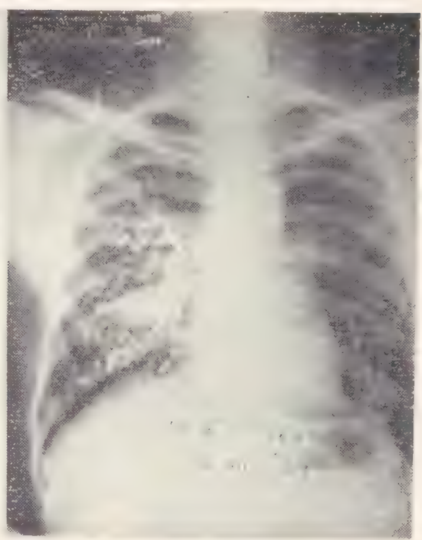


Fig. 2. — Case # 1

The first chest film (Fig. 1) taken in 1946 shows a shadow near the right hilum which was interpreted as "hilar type of Tb."

Figure 2 reproduces the bronchogram, done at San Patricio in 1949. This shows some bronchiectasis in the same area.

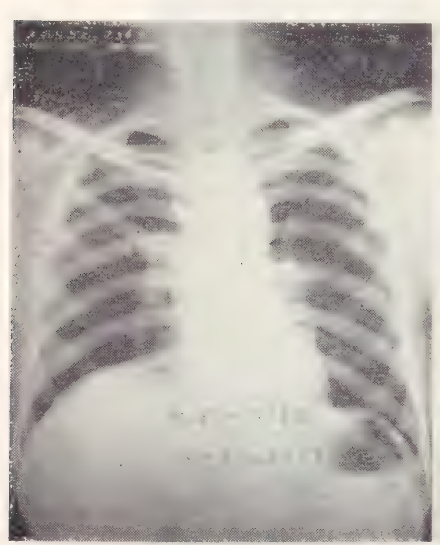


Fig. 3 — Case #1



Fig. 4 — Case #1

The chest film in 1952 (Fig. 3) shows no significant change. The last chest x-ray in 1955 (Fig. 4) reveals fluid at the right base. It must be remembered that the patient is now suffering from Laennec's cirrhosis. The original fibroid change near the right hilum is about the same.

Comment:

The fact remains that the clinical diagnosis of pulmonary Tb was established in 1946 on the basis of an erroneous x-ray report. This diagnosis remained solidly printed in every final summary of each hospital admission.

After having reviewed the records of all these 23 hospital admissions and numberless chest films, I venture to say that this man did not have Tb in 1946. He certainly had a good chance to contract Tb throughout these 9 years, living for long periods in Tb hospital wards. Actually, I would not be surprised, if one day he would develop Tb as a complication of his liver cirrhosis.

This case shows 2 mistakes: First, overreading of the film by the radiologist. Second, the clinician's blind and continuous acceptance of x-ray reports, in spite of persistently negative bacteriological studies.

Case #II

N. F. — Age: 64

This man was referred to our x-ray Dept. directly from the admitting room with the x-ray request slip stating: "hemoptysis since yesterday".

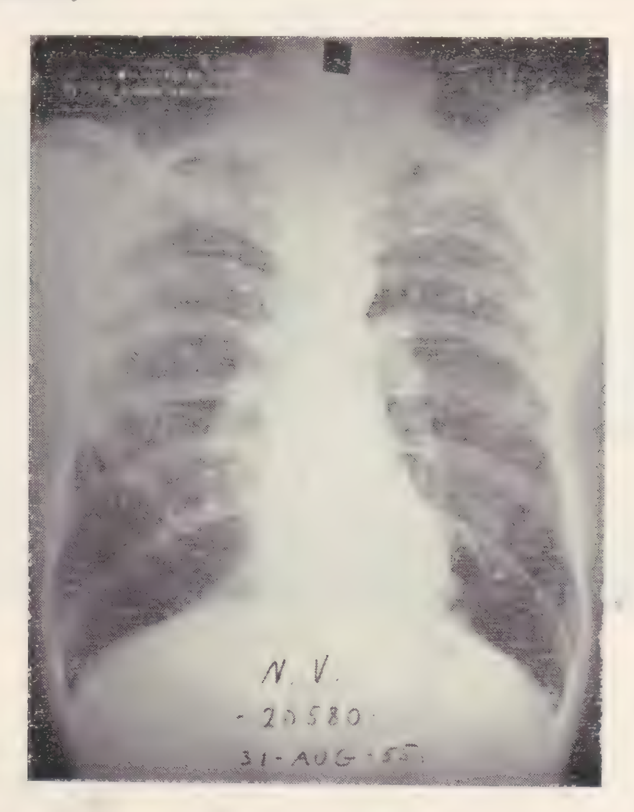


Fig. 5 — Case #2

The chest film (fig. 5) was reported as showing "emphysema and far-advanced Tb. with suggestion of cavity in the R.U.L.". The report also stated: "since emphysema with chr. bronchitis may produce similar appearance, the above impression (Tb) should be confirmed by sputum examinations".

The patient was immediately transferred to a Tb hospital. There, the sputum was negative for Tb. A bronchogram was ordered, however, the patient left the hospital against medical advice because he was placed in a ward with Tb patients. Follow-up has revealed no evidence of Tb.

Comment:

This is another case of overreading of a chest film by the radiologist and of hasty labeling of a patient by the clinician on the basis of an x-ray report.

Case #III

D. A. Age: 60

This patient had 9 San Patricio admissions.

On his 4th. admission, in March 1953 for anterior chest pain, the chest film (Fig. 6) showed a small translucency in the rt.



Fig. 6 — Case #3

clavicle, interpreted as a probable benign lesion; however, a biopsy was advised by the radiologist. X-ray examinations of the other bones were negative, except for some degenerative changes in the dorso-lumbar spine. Laboratory tests were within the normal limits. Patient was discharged with the clinical diagnosis of osteoarthritis.

Four weeks later, a special view of the upper dorsal spine was requested by the Orthopedic Service. This revealed collapse of D.2., due to a destructive process. X-ray impression: Giant cell tumor; myeloma should be ruled out. (I cannot show the slides, because it is difficult to see the lesion even on the original films.). On reviewing the films of the dorsal spine taken 4 weeks before, one could identify in retrospect some abnormality about the 2nd D. vertebra.

Interestingly, reexamination of the rt. clavicle showed marked regression of the lesion. In spite of this, a biopsy of this lesion was performed, and was reported myeloma by the Pathology Department.

There are other remarkable features in this case. Suffice to say that during the following 2 years the clinical picture was most unusual. All laboratory tests including several bone marrow examinations were negative. On his 9th. admission in 1955, the initial clinical impression was: "Psychoneurosis, rule out myeloma". The chest film this time showed complete healing at the site of the previous biopsy in the rt. clavicle. It showed, however, some minimal mottling in the left 9th. rib covering an area of about 1 cm. long. This area of the rib was resected and the pathology report revealed myeloma.

Comment:

In this case, the radiologist's initial diagnostic impression of a probable benign lesion of the right clavicle was wrong. His advice regarding biopsy was first ignored. Two years later, the clinical diagnosis was still in doubt. The correct x-ray interpretation of the minimal changes in the 9th. left rib led to prompt biopsy and confirmation of the pathological diagnosis of myeloma.

The last two cases will illustrate the radiologist's difficulties in arriving at a definite diagnostic impression and perhaps some insight into his anxieties when he is viewing chest films.

Case #IV
R. E. — Age: 64

This patient was admitted for a "swollen right testicle". The routine chest film showed a hilar mass on the right side. (Fig. 7).

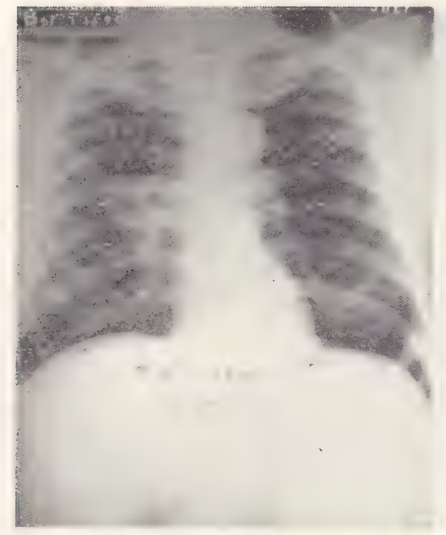


Fig. 7 — Case #IV

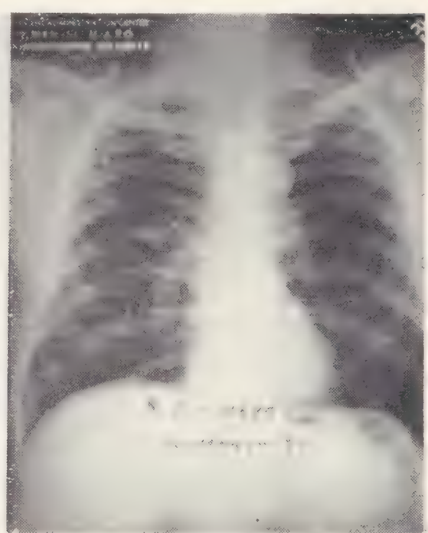


Fig. 8 — Case #IV

No interpretation was offered in the x-ray report. The clinician in charge of the case was personally informed, however, that CA was suspected.

Physical examination of the chest and all the laboratory tests were negative. Bronchoscopy revealed nothing unusual.

The chest film, 3 weeks later, (Fig. 8), was essentially clear.

Case #V
G. J. — Age: 59

This patient was admitted in Jan. 1955 with the diagnosis of chr. glomerulonephritis; the EKG revealed evidence of myocardial infarction. The routine chest film was reported as showing "a prominent left hilum presumably due to a dilated pulmonary artery. Two different radiologists noticed pulsations in the region in question.

Two months later, however, the patient developed bloody expectoration with pain in left chest associated with slight fever. He was referred to us with the clinical diagnosis of pulmonary infarct. The chest film showed increased lung markings at the left base and was reported, "as consistent with the clinical diagnosis of infarct."

Shortly after the patient became asymptomatic.

One month after, however, he had another episode of bloody expectoration. This time the chest film (May 1955) showed definite increase in the size of the left hilum and some fluid at the left base. (Fig. 10). The x-ray impression was bronchogenic Ca, L.L.L.

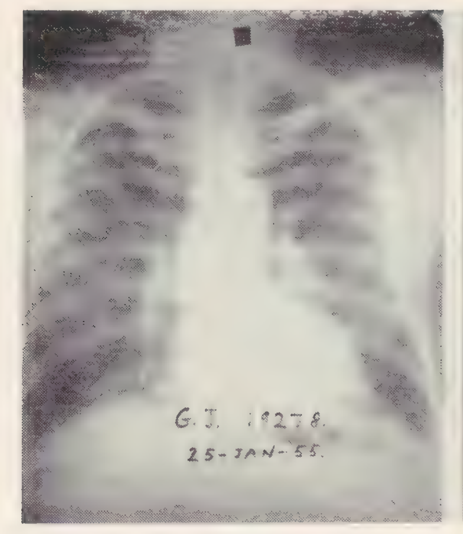


Fig. 9 — Case #V

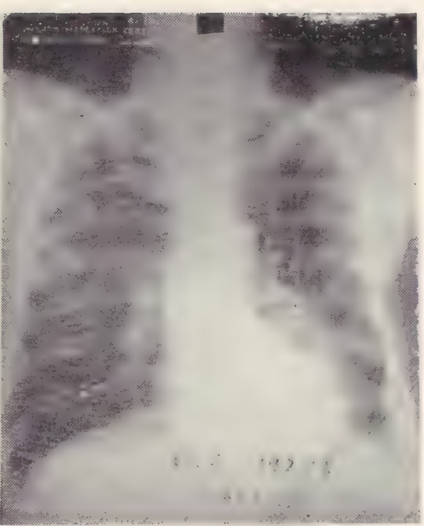


Fig. 10 — Case #V

This x-ray impression was quite a shock to the clinician in charge of the patient. Repeated bronchoscopies and biopsies reported anaplastic bronchogenic Ca.

This patient died 4 weeks later.

Comment:

These two cases illustrate the relative advantage of "over-reading".

The first case was overread. This stimulated the clinician into a thorough investigation which finally eliminated the possibility of carcinoma.

On the other hand, the second case was underread. This lulled the clinician into a false sense of security to the point that when the x-ray impression of Ca. was submitted, he refused to believe it until he had two positive biopsies.

CONCLUSION

In conclusion, I want to emphasize the original premises illustrated by these cases:

1st: The x-ray interpretation is a diagnostic impression. It is not a diagnosis.

2nd: The clinician has a right to expect a definite x-ray diagnostic impression.

3rd: The responsibility of the radiologist begins and ends by the correct interpretation of the x-ray findings, but the full responsibility of the final diagnosis rests with the clinician.

ELECTROLYTE CHANGES IN CONGESTIVE HEART FAILURE

*ELI A. RAMÍREZ-RODRÍGUEZ, M.D.,**

The clinical course of congestive heart failure is attended by frequent changes in electrolyte concentration. Many of these are mainly of physicochemical importance and have little clinical significance at the present time. This presentation is restricted to those electrolyte changes of congestive heart failure considered to represent important diagnostic or therapeutic problems.

These are not exceptional clinical situations. In San Patricio Hospital they have been recognized in an appreciable number of patients with congestive heart failure. This may be attributed to the fact that these patients are followed very closely with excellent diagnostic facilities from their first admission to the hospital and later course in the out patient cardiovascular clinic, to their very last stages of congestive deterioration and death. This brief review of the subject is a result of the interest elicited by the clinical problem as met during the management of these cases.

In dealing with electrolyte disturbances it must be kept in mind that the measured electrolyte concentration is a function not only of the actual electrolyte amount but also of the quantity of fluid. A given electrolyte concentration decrease may be due as much to actual electrolyte loss as to fluid gain, and conversely an electrolyte concentration increase may be due to actual electrolyte gain or to fluid loss. While electrolyte disturbances may predominate in single ions they should be considered to affect total ionic as well as water balance.

Etiology

Fluid-electrolyte disturbances occur in heart failure because of one or more of three fundamental mechanisms.

The first of these is the disturbed pathologic physiology which follows the congestive state itself. This disturbance is characterized by retention of both water and solute, but there is predominant retention of water leading to electrolyte dilution in spite of increased electrolyte stores. In other words, in the usual congestive heart failure there is a decrease in measured electrolyte concentration which is due to an excess of water retention over solute

* Chief, Medical Service, San Patricio Hospital.

as distinguished from actual electrolyte loss. It is important to recognize this state of slight electrolyte dilution as the baseline reference from which additional electrolyte changes are to be gauged.

The second mechanism for electrolyte disturbance in heart failure is diuretic therapy. Whether elicited by dietary restrictions or by specific agents, diuresis depends on considerable fluid electrolyte shifts in order to reduce congestion effectively. Ordinarily there follows rapid restoration of fluid electrolyte balance to a level compatible with normal cellular metabolism depending on the integrity of compensatory adjustments.

Impairment of compensatory adjustments (primarily renal), is the third mechanism which contributes to or produces fluid electrolyte disturbances in heart failure. In some patients, particularly elderly arteriosclerotic individuals, such renal dysfunction may be undetectable by the usual clinical tests; nevertheless evidence of abnormal renal function should be sought in all cases of congestive heart failure in order to appraise this aspect of fluid-electrolyte disturbance risk. Electrolyte depletion itself may magnify or even cause renal insufficiency setting up a vicious cycle of increasing renal insufficiency, caused by and causing further electrolyte disturbance, and leading to eventual irreversibility.

Clinical Features and Treatment (Fig. 1 and 2)

Hypochloremia

The most common electrolyte depletion of congestive heart failure is predominant hypochloremia. The usual history is of relatively mild cardiac failure which has responded vigorously to mercurial diuretics and suddenly loses mercurial response, sometimes in spite of edema. These patients frequently have weakness and muscular cramps. If excessive water is lost there may be dehydration also. The serum chloride is markedly decreased and the carbonic ion increased with the production of alkalosis. Significant nitrogen retention appears late.

The treatment is 6.0 gms. of ammonium chloride per day in capsules, solution or enteric coated tablets. If necessary it may be given intravenously in 2.14% solution, not to exceed 200 cc. per hour or 1 to 1½ liters per day. These limitations are imposed by the danger of ammonium ion intoxication, which may be forbidden in cases of hepatic or renal dysfunction. In habitual heavy chloride losers hypochloremia may be prevented by oral ammonium chloride before and during mercurial effect.

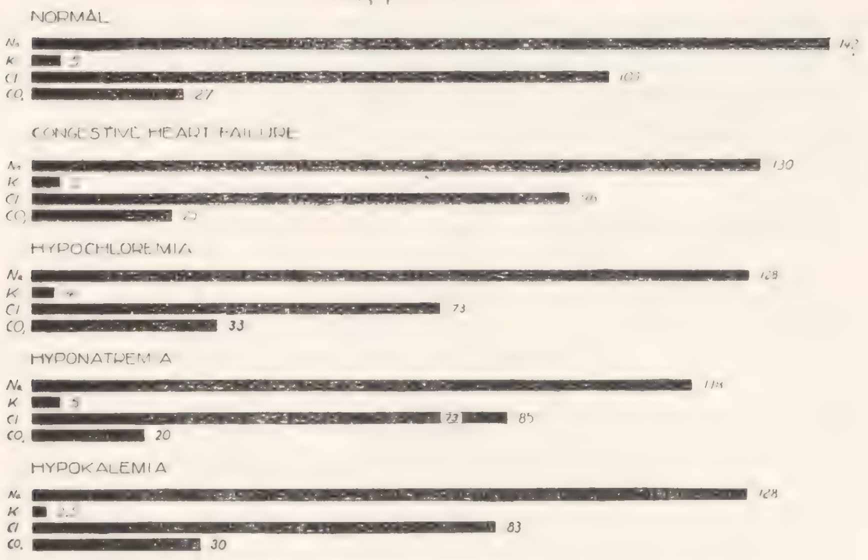


Fig. 1. Electrolyte patterns in congestive heart failure.

	HYPOCHLOREMIA	HYPONATREMIA (Depletion)	HYPONATREMIA (Dilution)	HYPOKALEMIA
Main Clinical Features	Cramps Mercury Block	Dehydration	Congestion	Shock-like state Arrhythmias
Incidence	Relatively common	Relatively uncommon	Probably common as terminal event	Probably common
Usual History	Mild cardiac with good Hg response which suddenly stops	Moderate cardiac with fair response to energetic diuretic treatment which is gradually lost	Bad cardiac, no apparent etiologic factor	Other electrolyte disturbances; diuretics; (Digitalis Intoxication)
Edema	Usually none	Usually none	Marked	May or may not
Serum Sodium	Normal or slightly increased	Markedly decreased	Decreased	Normal
Serum Chloride	Markedly decreased	Slightly or moderately decreased	Decreased	Decreased
CO_2 Comb. Power	Increased (Alkalosis)	Decreased (Acidosis)	Decreased	Increased (Alkalosis)
NPN	Normal till late	Elevated early	Mild to moderate elevation	May be elevated (renal insufficiency)
Treatment	Chloride	Sodium (Chloride if needed)	Improve myocardial function	Potassium
Prognosis	Good	Fair if treated early	Ominous	Fair to good if treated early, depending on cause

Fig. 2. Clinical features of electrolyte disturbances of congestive heart failure.

Hyponatremia

Hyponatremia in congestive heart failure may be of two types. One is due to actual sodium loss and is called depletion hyponatremia. In the other type the diminished sodium concentration is part of a generalized electrolyte dilution due to excessive water retention. This is called dilution hyponatremia.

Depletion Hyponatremia

Predominant depletion hyponatremia is relatively uncommon. It may follow the use of Diamox or cation exchange resins, or may be due to prolonged diarrhea. Combined hyponatremia and hypochloremia may occur after frequent mercurial diuresis, after excessive sweating, or after removal of salt containing fluid collections (such as paracentesis, thoracentesis, gastrointestinal drainage, Southey tubes, etc.) followed by replacement of water only. These factors may be enhanced by drastic sodium chloride restriction. The usual history is of moderate cardiac failure which has responded fairly well to energetic diuretic measures and gradually loses diuretic sensitivity, sometimes in spite of persistent edema. These patients are usually dehydrated, manifesting weakness, loss of skin turgor, drowsiness, apathy and hypotension. The serum sodium is low, the chlorides low or normal, and the carbonic ion is low with the production of acidosis. There are oliguria and early nitrogen retention.

The treatment is sodium, plus chloride and water as needed. Loss of sodium alone may be corrected with oral sodium lactate or gluconate. If necessary it may be given intravenously as sodium lactate in more or less concentrated solution according to water loss. Loss of sodium and chloride in excess of water may be corrected by withholding water and giving salt; preferably in an absorbable palatable vehicle such as soup, rather than in tablets. If necessary it may be given intravenously in more or less concentrated solution according to water loss. The amount of sodium chloride needed can be estimated by multiplying total body water (50-70% of body weight) in kilograms times the measured deficit in grams per liter. Not more than one-third of the figure obtained should be administered in a twenty-four hour period at which time a redetermination of ionic concentration should be done before further electrolyte treatment. A single intravenous administration of 5% salt solution should not exceed 200-250 c.c.; the maximum in twenty-four hours is 400-500 cc. These limitations are imposed by the danger of rapid plasma volume expansion and subsequent pulmonary edema.[2]

Dilution Hyponatremia

In the second form of hyponatremia the decrease in plasma sodium concentration is not due to sodium loss but to excessive water retention in spite of normal or increased electrolyte stores. As already described a similar change is characteristic of the congestive state, but in this so called dilution hyponatremia it becomes markedly exaggerated. This disturbance is not exclusive to heart failure as it may be seen also in advanced renal failure and cirrhosis of the liver.[4] These patients develop in effect a new electrolyte balance with isotonicity set at a very dilute level. The definite cause of this phenomenon is unknown although several possible mechanisms have been suggested.[4]

The clinical picture is of advanced congestion and edema. There is usually no sign or symptom referable to electrolyte disturbance despite the hyponatremia and low urinary sodium. The disturbance is probably common as a terminal event in congestive heart failure and when it appears the prognosis is ominous.

The differential diagnosis between depletion and dilution hyponatremia is difficult to establish. The presence of edema and a gradual development of hyponatremia without resort to a significant etiological factor (such as diuretics) favor the dilution type, but are not conclusive. Best differentiation can be established with serial extracellular fluid volume determination but unfortunately there is no satisfactory method of practical clinical application available at the present time.[6]

The treatment of dilution hyponatremia in congestive heart failure is the correction of the myocardial insufficiency. Inasmuch as it is so difficult to rule out depletion hyponatremia, a cautious trial of hypertonic sodium chloride is advisable. If this fails in improving the patient's clinical condition and or the plasma sodium concentration, more sodium chloride is contraindicated. Obviously if the problem is dilution and the sodium stores are normal or increased, hypertonic saline is not indicated and may be actually harmful.[3] Recently ACTH has been used experimentally to produce water diuresis in some of these cases.[5] Further investigation of this novel and paradoxical approach appears to be in order before general clinical application.

Transitory dilution hyponatremia can occur in cardiac patients when they are given large amounts of saltless water or when they become water-logged in anuria. It has been reported also during the post-operative period in patients with congestive heart failure who undergo successful mitral commisurotomy.[7]

Hypokalemia

In contrast to hyperkalemia, which is not observed unless there is marked renal insufficiency or overly enthusiastic potassium therapy, hypokalemia is relatively common in heart failure. Potassium changes affect both intracellular and extracellular osmolarities because of its distribution in both fluid compartments. In potassium depletion the extracellular and myocardial intracellular deficits must be defined separately because they may not occur concurrently and their manifestations differ.

Extracellular potassium depletion may be due to diuretic agents such as Diamox, cation exchange resins (particularly when administered in their hydrogen cycle only) and mercurials. The hypokalemic action of these agents may be enhanced by diarrhea. It is very important to remember that hypokalemia may appear also during recovery from hyponatremia because of: (a) intracellular replacement of potassium by sodium, (b) potassium flushing by diuresis and (c) plasma volume expansion by hypertonic solutions. Hypokalemia manifests clinically as a shock-like state with muscular weakness, rapid pulse, shallow respirations, "fish-mouth" expression, muscle paralysis, collapse and death. Besides the low potassium concentration the plasma shows hypochloremia and alkalosis. Prolonged hypokalemia may produce renal failure with uremia because of renal damage characterized by vacuolization and hydropic degeneration of tubular cells.

Myocardial cellular potassium depletion usually follows hypokalemia. However, the serum potassium concentration may not reflect at all times the cellular potassium level because of characteristic myocardial avidity for potassium and because of delay in establishing ionic equilibrium between the intracellular and extracellular compartments.[6] Animal experiments have shown that myocardial potassium depletion may be due also to digitalis poisoning. Conversely, myocardial potassium depletion may induce cardiotoxic manifestations to digitalis in subjects well stabilized at a fixed maintenance dosage.[8] Indeed, the arrythmias appearing after effective diuresis in digitalized patients are probably caused by increased digitalis sensitivity due to myocardial potassium depletion rather than by digitalis mobilization as was believed formerly. This digitalis-potassium relationship is the basis for the recommended routine use of potassium in the treatment of digitalis intoxication. Myocardial potassium depletion is manifested by certain semi-specific electrocardiographic changes. As in digitalis effect there are ST depression of the spoon-shaped type and T wave inversion but the QT interval is lengthened instead of shortened. Premature auricular and ventricular beats, auricular tachy-

cardia and other arrythmias may occur, particularly during digitalis administration.

For myocardial potassium depletion and hypokalemia the treatment is one to two grams of potassium citrate given in orange juice three or four times a day. If there is hypochloremia, potassium chloride may be given instead. When and if intravenous administration is necessary, the ability of the kidney to eliminate the potassium excess must be evaluated. With normal renal function up to 20 mEq. may be given safely in a four hour period.

CONCLUSIONS

The following recommendations are suggested in order to help prevent the electrolyte disturbances of congestive heart failure:

1. The digitalis, diuretic and salt restriction requirements of the patient with congestive heart failure must be strictly individualized according to specific indications.

2. In every cardiac requiring treatment for congestive failure the status of renal function should be evaluated in order to help establish the electrolyte disturbance risk.

3. Suitable chemical and electrocardiographic laboratory facilities should be available for diagnosis and control of treatment.

4. Electrolyte depleting factors such as diarrhea, vomiting and excessive sweating (the latter especially in Puerto Rico) should not be neglected; least of all in cardiac patients who are most susceptibe to them.

5. The routine administration of four glasses of fresh orange juice on the day of mercurial diuresis is recommended for prevention of hypokalemia.

6. The habitual heavy chloride loser should be given six grams of ammonium chloride the day before and the day of mercurial diuresis to prevent hypochloremia.

7. Hypokalemia should be suspected always as a possible background complication of other electrolyte disturbances.

8. There is considered to be enough occumulated evidence to recommend the routine use of potassium in the treatment of digitalis intoxication unless specifically contraindicated.

9. A high level of clinical awareness of the possibility of electrolyte changes in congestive heart failure must be maintained.

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PREVENTION OF SHOULDER DEFORMITY*

*HERMAN J. FLAX, M.D., M. Med. Sc. (Phys. Med.)***

The usual explanation for the production of stiff and painful shoulder is disuse, secondary to prolonged immobilization. The original lesion in my experience is usually painful calcific deposits in the short rotator tendons, trauma to the upper extremity or paralysis of the shoulder muscles. Since entering private practice, the problem of periarthritic personality[1-2] in prolonging shoulder deformity has become more and more prominent. Unless this factor is recognized and seriously considered by the physiatrist, the treatment regime will fail.

The great number of possible causes[1-3] of stiff and painful shoulder calls for a correct diagnosis in order to prescribe treatment and evaluate prognosis. The internist or orthopedist must determine whether the patient is to be treated conservatively or by surgery. Nevertheless, complete roentgenographic examination is necessary in order to reach a correct diagnosis. Once this diagnosis is determined, and the selected treatment is instituted, the need for early mobilization to prevent the unwanted complication of a "frozen" shoulder is mandatory.

The following method of treatment is based on the work of Inman, Saunders and Abbot[4] who studied the action of the various muscles of the musculotendinous cuff in maintaining the balance of force in shoulder motion. There is first a combination of forces exerted by the short rotators on the humeral head to fix it in the glenoid cavity, after which time the longer and stronger muscles of the shoulder take over the movements. Accordingly, treatment is directed primarily to mobilizing and strengthening the short rotator muscles.

Examination of the Patient:

First, range of joint motion is determined (Fig. 1), and whether the limitation of abduction is caused by an intraarticular or extra-articular lesion. Next, strength of the shoulder girdle muscles is determined. Finally, each muscle of the shoulder and neck is palpated to determine the presence of pain and localized spastic areas.

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** Chief Physical Medicine and Rehabilitation Service, San Patricio VA Hospital, San Juan, Puerto Rico.
Assistant Clinical Professor, Physical Medicine and Rehabilitation, University of Puerto Rico School of Medicine, San Juan, Puerto Rico.

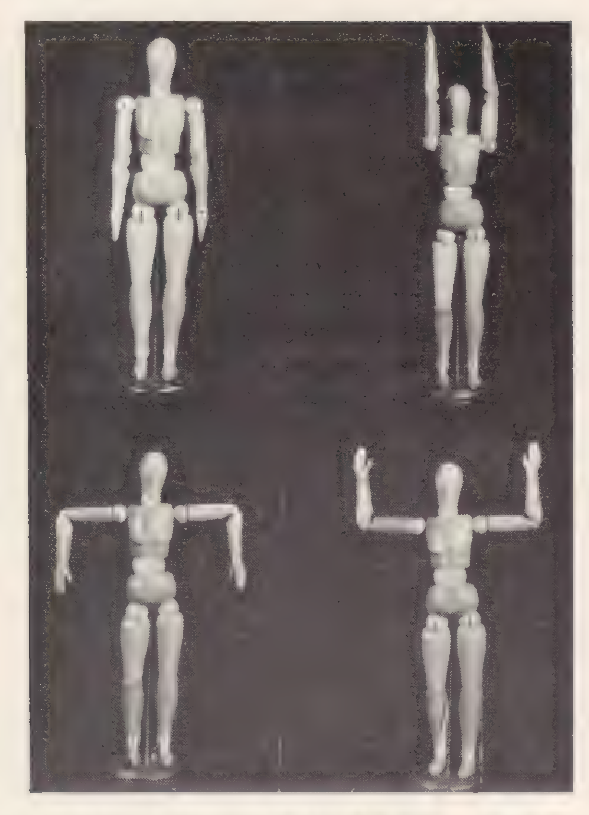


Figure 1. Manikin showing complete range of motion: Abduction 0-180 degrees. Internal Rotation, 0-90 degrees. External Rotation, 0-90 degrees.

The range of internal and external rotation is measured with the arm abducted to 90 degrees if possible. Any limitation of abduction and rotation, either external or internal or both, denotes an intrarticular cause, such as acute peritendinitis calcarea (Fig. 2). Limitation of abduction with full range of rotation suggests an extraarticular cause, such as myositis following thoracotomy or paralysis of shoulder muscles (Fig. 3). It is very common to find disuse muscular atrophy with capsular and pericapsular fibrosis complicating extraarticular lesions. However, the presenting picture is interference with rotation and cannot be distinguished from a primary intraarticular cause.

The strength of the shoulder muscles is determined, unless the area is too painful to be moved. If there is any doubt about a peripheral nerve lesion, a Reaction of Degeneration determination is done. Otherwise, no further attention is paid to muscle strength until progressive resistive exercises are started.

A comfortable position is necessary before the muscles are

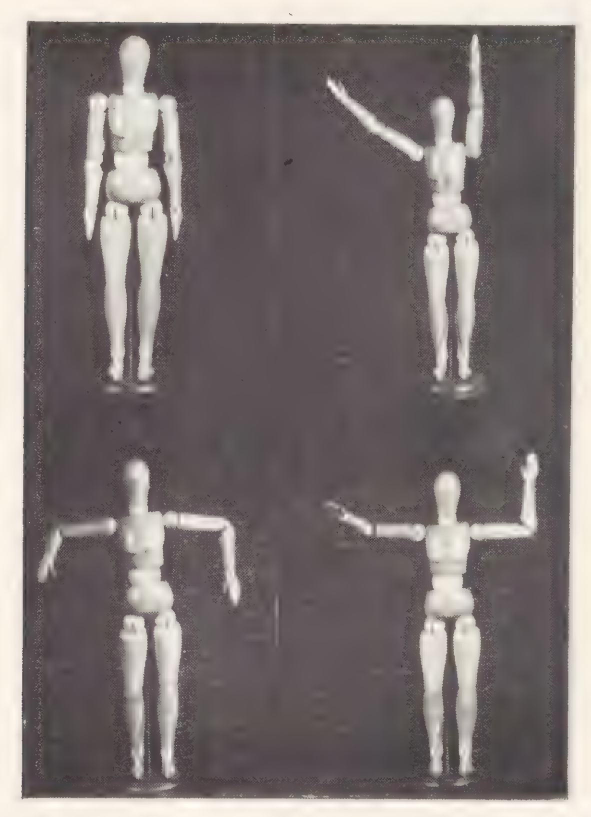


Figure 2. Manikin showing intraarticular lesion: Abduction is limited, Internal and External Rotation are limited.

palpated. The patient lies prone on the treatment table, pillows are placed under the chest and feet, the upper extremities hang over the sides and he rests on the forehead. Diagnostic massage of each muscle group, following Storm's Technique[5] is done. In "acute" involvement, the shoulder muscles, especially their scapular attachments, reveal spastic and tender areas. The patient winces invariably with pain when they are palpated. In the "chronic" shoulder not as much spasm is felt, but, instead, painful, indurated, "fibrotic" areas are found usually in the muscle bodies.

The neck is examined in the supine position with a firm pillow under the head. In practically every patient muscle spasm is palpated in the cervical paravertebral, occipital and trapezius muscles on the affected side. These areas are thickened, firmer and exquisitely tender as compared to the surrounding tissue. This finding is so constant in acute shoulder involvement, that if not felt, the severity of the lesion is minimized.

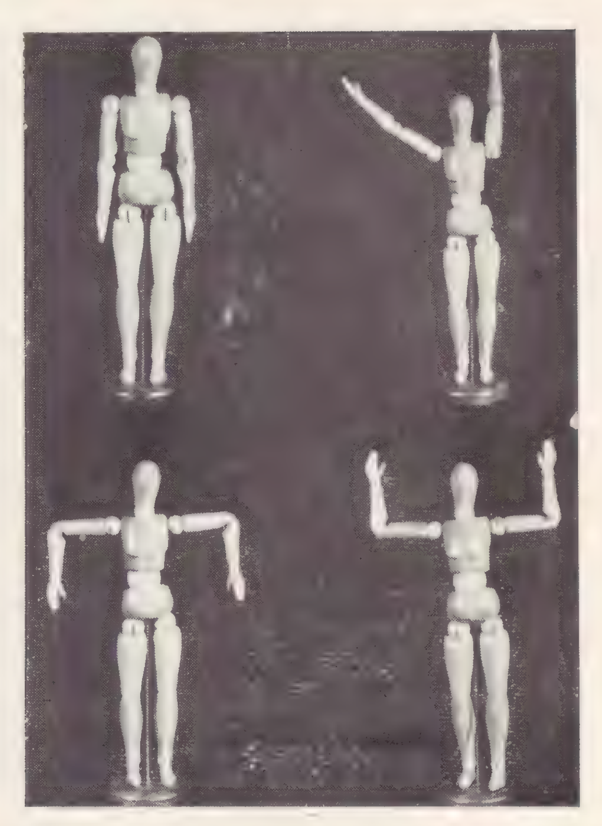


Figure 3. Manikin showing extraarticular lesion: Abduction is limited, Internal and External Rotation are normal.

Treatment of Acute Shoulder Lesion:
Primary Treatment:

During the first 48 hours following the onset of an acute peritendinitis calcarea, the shoulder and the patient are kept at complete rest. Medication in adequate doses of narcotics and sedatives, prednisone and salicylates is prescribed.

The affected shoulder is supported by placing the extremity in a large triangular bandage. This, in itself, relieves pain by preventing the constant pull of gravity and arm motion on the inflammed tissues. Normally, the patient is put to bed, and immobilization is maintained by means of supporting pillows. A large, pliable, plastic, ice bag is applied continuously against and around the shoulder. The analgesic value of cold can be attested by the fact that most patients require such less sedation when ice is continuously applied to the involved shoulder. Of great importance, from the standpoint of future treatment, is the need to assure and

reassure the patient that the complication of a "frozen" shoulder will not occur with proper treatment. Nothing more is done until the acute pain is relieved, usually after 24 to 48 hours of rest, medication and assurance.

The use of surgical procedures, such as needling and incisions, injections of local anesthetics and hydrocortisone or roentgen ray therapy to relieve the symptoms of an acute shoulder lesion, have not been necessary in my experience. Since the introduction of cortisone and prednisone, the relief of pain has been so spectacular that these procedures, most probably, will be used only rarely in the future.

Medication:

Medication is continued in decreasing quantities except for the salicylates, which are given in 10 grain enteric coated Sodium Salicylate tablets, 20 grains, 4 times daily for 7 to 10 days, unless symptoms of salicylism are produced. With very few exceptions, after 48 hours, Demerol (R), 100 milligrams intramuscularly every 4 hours, is discontinued, and Butisol (R) Sodium, $1\frac{1}{2}$ grains every 4 hours, is cut down to one tablet at bed-time.

Meticorten (Prednisone, Schering) is rapidly diminished, once the acute pain is relieved, usually from 24-48 hours following onset of medication. The prescribed quantity is 10 milligrams 4 times daily for 2 days, 10 milligrams twice daily for 2 days, 5 milligrams twice daily for 2 days and finally 5 milligrams daily for 2 days. The total amount of meticorten (R) taken rarely exceeds 210 milligrams over a 10 day period. All medication is discontinued usually by the tenth day, but the patient continue on physical therapy.

Physical Therapy:

Acute inflammatory lesions of the shoulder are self limiting, but immobilization over a prolonged period will result in permanent stiffness. In order to prevent this complication, after 48 hours of rest, a dynamic daily program of physical therapy is prescribed. Micro-wave or short-wave diathermy to the neck and shoulder regions for 30 minutes, followed by diagnostic massage,[5] Sayre sling head traction, active non-resistive exercises, gentle active and passive manipulation and constant reassurance is instituted. Head traction, using 40 to 60 pounds of pull for 3 to 5 minutes, is used routinely since most of the patients present painful areas on massage of the cervical paravertebral and occipital muscles, even with-

out demonstrable vertebral pathology. Stretching also relieves the tight, "tired" feeling in their necks and adds to their comfort.

Massage:

Massage is continued until all the tender, spastic areas in the muscles have disappeared. It is a common finding in patients with "chronic" shoulder limitation to find firm, indurated and exquisitely tender areas in the latissimus dorsi, teres major and pectoralis major muscles. These are the adductors of the humerus[6] and any pull on these painful areas sets up reflex muscle splinting. The supraspinatus and deltoid muscles must be carefully "ironed out" by massage to facilitate abduction. Each and every muscle of the shoulder girdle must be carefully palpated by means of diagnostic massage[7] because a tender, spastic area in any of these muscles may be the cause of prolonged shoulder pain and subsequent limitation of movement. On occasions, and this is more often true in the "chronic" shoulder lesions, these painful points may have to be anesthetized by local infiltration with one per cent Novocain (R) Solution before massage can be attempted.

Active Exercises:

Active exercises are introduced at the very onset of physical therapy in the form of pendulum, pulley and simple rotation movements. To do pendulum exercises, the patient may stand, sit or lie prone with the affected arm dangling beside the table. Usually, the patient stands and bends over, allowing gravity to carry the extremity forward. He remains in this bowed position without moving the elbow and moves the arm first vertically, then horizontally, next clockwise and finally counterclockwise in an ever increasing arc within the limitations of pain tolerance. As soon as the maximum range of motion is reached, the arc is dimished slowly to restig position.

An overhead pulley system can be constructed for home use by fastening a single one-inch pulley to an overhanging door frame. A length of rope is passed through the pulley and looped at either end. Each hand grasps a loop, and the involved extremity is forward flexed by pulling down with the other. In due time, resistance can be offered by the uninvolved extremity. This is an excellent therapeutic exercise.

"Back wiping" is good for both internal and external rotation. The elbow must be kept immobile, otherwise the head of the

humerus will not rotate within the glenoid cavity. The patient grasps the lower pole of a towel (or rope) with the hand of the affected side, supinated behind the back. The other hand pulls the towel up across the opposite shoulder as far as possible. Next. the hands are reversed, and the towel is pulled down behind the back as far as possible.

Another exercise is for the patient to stand straight, heels, buttocks, shoulders and head touching the wall, and place both hands behind the neck (or as far back as possible). The patient tries to touch the elbows back against the wall and then together in front of the face. This exercise is for external rotation.

Wall climbing is an excellent exercise for flexion and abduction, as well as to measure daily progress. The patient stands facing the wall and climbs step-ladder fashion up and down the wall with his fingers. A mark is scored as the highest point of flexion reached. Then, the patient turns 90 degrees with the involved side facing the wall and attempts the same maneuver. He is cautioned not to twist his body forward but to remain sideways. A mark is made at the height of abduction. The patient is stimulated to compete against himself and to improve his record with each attempt.

These simple exercises constitute the program practiced at home. They are done four times daily to tolerance. The rotation exercises are continued "ad infinitum" once or twice daily through complete range of motion. Maintaining full range of motion is an added insurance that acute shoulder pain will not reoccur unless there is some untoward change in the pathology of the lesion.

Resistive Exercises:

Progressive resistive exercises are started as soon as the acute pain and muscle spasm disappear. An adjustable see-saw apparatus (Fig. 4) is used to strengthen the short rotators as well as the longer shoulder muscles. The patient sits in an ordinary straight-back chair, and his affected arm is strapped to the cross bar with the elbow bent to a right angle. The arm should be abducted to 90 degrees, but if this is not possible, the apparatus can be adapted to the desired height. The arm is raised and lowered, gradually increasing the arc of movement. As soon as full range of painless motion is achieved, weights are added to either end following the DeLorme's technique of progressive resistive exercises[7]. As soon as the patient is able to lift a weight approximately comparable to that raised by the non-affected side, these exer-



Figure 4. Adjustable See-saw Apparatus for Progressive Resistive Exercises. Modified from equipment seen at Workmen's Compensation Center, Malton, Ontario, Canada, and Sunnybrook Veterans Hospital, Toronto, Canada.

cises are discontinued. This takes usually from two to three weeks in acute intraarticular lesions.

Resistive as well as active non-resistive exercises must be used with caution. Any complaint of increased pain the next day is a sure sign that too much has been done, and the exercise program must be stepped down. The zeal to build muscles must be tempered in order to avoid destroying muscle.

This is especially true in the postoperative course following chest surgery. Thoracoplasty and thoracotomy operation are no contraindication to see-saw resistive exercises if done within the patient's tolerance, influence no change in temperature, pulse and respiratory rate and produce no palpable spasm in the shoulder muscles. This technique of resistive exercises is very valuable to treat the stiff shoulder resulting from adhesive capsulitis, periarthritis and bicipital tendinitis.

Manual Manipulation:

Manual manipulation is rarely necessary in the treatment of acute shoulder lesions. On the other hand, it is of great importance in the stiff shoulder after all other methods, including mechanical manipulation on the see-saw device, have failed. Whenever necessary, and only once a week, the shoulder joint is injected with 10 cubic centimeters of one per cent Novocain (R) Solution and 50 milligrams of Hydrocortisone, after which gentle but forceful manipulation following the technique of Mennell[8] is done. With only few exceptions, the patients have reported progress following this procedure. It is important that the scapula as well as the humerus is manipulated.

End Results:

The usual length of time for patients to recover full range of painless motion following an episode of acute peritendinitis calcarea is 10 days. The normal duration of treatment for a partially fibrosed shoulder is five weeks. This is also the prevailing period necessary to promote complete shoulder motion following chest operation if treatment is started immediately after surgery. There is no specific time for treating the stiff shoulder. Anywhere from three to nine months has been my experience. This depends on the "will to recover"[1] and the 'reasons for shoulder pain causing periarthritis"[1], of which the periarthritic personality[1,2] reflex sympathetic dystrophy states[9,10] and compensation claims[11] are but a few.

The end result is measured not only by full range of humeral movements, but also by full range of scapular movements. This can be determined easily by drawing a line around the vertebral border and inferior angle of both scapulae, first at rest and then in complete abduction. (Fig. 5). The difference between two lines should be about the same before the patient is discharged. This measurement is of special value in the selection of patients for discharge following chest surgery. Unless the scapula is completely mobilized, there will be limitation of shoulder motion due to scarring and contracture of the scapular muscles. (Fig. 6).

SUMMARY

The following treatment of acute shoulder lesions has proven effective in preventing deformity:

1. Complete rest of the patient and the shoulder for the first 48 hours. This is achieved with sedation, meticortene (R) and salicylates in proper doses, a supportive triangular bandage to the



Figure 5. S. R. S., Clínica Fernández García, April 12, 1955. Ten weeks following wedge resection of right upper lung. Full range of scapular motion. Complete range of shoulder movements.

shoulder, continuous application of an ice bag, a comfortable bed and reassurance.

2. Then microwave or shortwave diathermy locally, diagnostic massage[5] to eliminate the tender, spastic muscle areas and active exercises are started.

3. Progressive resistive exercise, using a special see-saw device, is instituted as soon as acute shoulder pain and muscle spasm disappear.

4. Manual manipulation is used only in those patients who do not progress with the previously mentioned treatment. It is used rarely in acute shoulder lesions but very frequently in stiff shoulders. Previous to manipulation, the shoulder joint is injected with 10 cubic centimeters of one per cent Novocain (R) Solution and 50 milligrams of Hydrocortisone.

5. The role of the psyche must be carefully evaluated as it is an important factor in prolonging shoulder disability. The patient must be reassured at all times that he will not develop a stiff



Figure 6. D. C., Clínica Fernández García, April 12, 1955. Eight weeks following decortication for chronic empyema left lung. Limitation of abduction despite complete rotation. Loss of full scapular motion secondary to scarring of vertebral border muscles.

shoulder and stimulated to exercise continuously. Otherwise, limitation of shoulder motion will persist.

6. The successful end result can be measured only by complete range of humeral and scapular motion. Scapular motion can be measured by outlining the vertebral border and inferior angle of both scapulae, at rest and complete abduction. The difference between these two measurements should be the same on both sides.

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SURGICAL CONFERENCE

SCHISTOSOMA MANSONI GRANULOMA WITH ABSCESS FORMATION, PARACOLIC, ADHERENT TO THE ASCENDING COLON AND ANTERIOR ABDOMINAL WALL;

REPORT OF A CASE

*LUIS A. PASSALACQUA, M.D., F.A.C.S.**
*RAFAEL RODRIGUEZ-MOLINA, M.D., F.A.C.P.***
*PEDRO J. OTERO, M.D.****

Perforation of the intestine by Schistosoma mansoni is rare in the medical literature. We have not been able to find a similar case. Manson's schistosome has been known to cause acute and chronic appendicitis,[1,2] acute appendicitis with strangulation of the ileum,[3] anorectal conditions,[4] cholecystitis,[5] pyelophlebitis of the liver,[6] hepatic abscess,[7]; it has been known to complicate a volvulus of the sigmoid colon,[8] and it is known to cause a variety of cardiac, genito-urinary and nervous conditions.[9,15]

In order to facilitate an understanding of the pathological process that distinguish this case, it seems well to discuss certain aspects of the life cycle of the Schistosoma mansoni in the snail and in man. Copulation of the adult male and female parasites takes place in the larger venous tributaries of the portal system. The gravid female then advances against the blood stream into the smallest visceral venous branches of the hemorrhoidal plexus of veins draining the descending colon and rectum. It is in these venules that the female worn deposits the eggs one by one. From here the ovum extrudes into the lumen of the rectum and colon and becomes incorporated into the feces, thus finding its way to the exterior of the human body. Numerous ova also are swept back into the portal circulation and liver, and here they remain as foreign bodies, giving rise to granulomatous lesions in the large intestine and in the liver. It should be mentioned here that the actual process by which the ova pass from the venules thru the vessel wall and into the tissues of the submucosa to the mucosa and into the free lumen of the intestine is controversial; however, the

* Chief, Surgical Service, VA San Patricio Hospital, San Juan, P. R.
** Chief, Medical Service, VA San Patricio Hospital, San Juan, P. R.
*** Resident physician in Surgery, VA San Patricio Hospital, San Juan, P. R.

NOTE: The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

lateral spine which is characteristic of the ova of Schistosoma mansoni may play a role in the mechanism of extrusion.

After passage from the intestinal tract of man the ova that come in contact with fresh water hatch into a larval form called miracidium. These penetrate certain species of snails and by a process of parthenogenesis, after three or four weeks, give rise to another larval form of the worm which is known as the cercaria. Fork-tailed cercariae emerge from the snail and infest the water. When a person wades or bathes in the water, the cercariae penetrate the skin and the tail drops off. This marks the beginning of what is known as the intracorporeal phase of the life cycle. From here the cercariae are carried by the blood stream, pass to the lungs and pulmonary circulation returning again to the heart and to all organs and tissues. These worms, however, exhibit a preference for the portal circulation and it is here that maturity is attained; the cercariae becoming adult male and female parasites within a period of about six to eight weeks after penetration of the skin. Other phases of the intracorporeal life cycle have already been discussed. With such an important habitat as the portal system of the liver, one can readily understand the seriousness and far reaching effects of the pathological processes that occur in man as result of infection with these parasites.

CASE PRESENTATION

Dr. L: This 40 year old white male was admitted to San Patricio Hospital on March 18, 1954 as a transfer from another institution. The patient gave the history of having developed epigastric burning pain on empty stomach and at night relieved by meals, back in 1942. At that time he was studied at an Army Hospital elsewhere and was found to have x-ray evidence of duodenal ulcer, and he was discharged from the Army on CDD. The patient continued to feel fairly well without following any diet, although occasionally he suffered from what he described as very severe constipation, having no stools for several days to weeks at a time. By 1948 he still complained of constipation and suffered an undetermined episode of severe upper abdominal pain. About four weeks before this current admission the patient had another episode of upper abdominal pain which lasted several days. On february 26, 1954 the patient suffered an excruciating upper abdominal pain for which he had to be hospitalized elsewhere. At this place he was found to have evidence of hypotension, BP 70 40, Temperature 97; Pulse 140; respirations of 40. The patient stated that he had very sticky perspiration and that his abdomen felt like

"cement" according to his words. He was very sick for several days but didt not receive blood or plasma or digitalis, but he did receive antibiotics. After some improvement he was transferred to our hospital. On the transfer notes we found that he had a white blood cell count of 41,000 per c.m.m. two days after the episode of the acute abdomen and 27,000 four days later. No mention of fever was made at that time. The patient said he had lost consciousness when he was sick at the other hospital. There was no history of schistosomiasis although he used tc go river wading in his hometown of Utuado, the last time he had been there being about two months prior to current admission.

On physical examination the patient was found to be undernourished and obviously dehydrated. The most important findings were referred to the abdomen, revealing marked resistance specially in the upper abdomen and more so on the left upper quadrant. Examination was unsatisfactory because this marked resistance persisted for many days. Several days after admission to the hospital, abdominal examination revealed that the condition was different and there was no resistance as it was previously found but rather suggestion of a mass on the left upper quadrant. The mass had been described by the various examiners but in general there had been agreement as to its movement both up and down on respiration and also from pressure behind the lumbar region.

Radiographic reports

Chest — essentially negative. **Upper GI** - there is definite evidence of a deformity on the anterior wall of the antrum of the stomach and on the greater curvature aspect of the distal half. Oblique projections and lateral views show that the distal 2/3 of the stomach are displaced medially and posteriorly. Careful study of the mucosal pattern shows this to be intact. **Summary:** definite evidence of extrinsic pressure at the distal 2 3 of the stomach displacing this organ medially and posteriorly. The stomach is displaced by a large mass. Further studies including small intestinal series, barium enema, I.V.P. will be done in an effort to determine the nature of this tumor mass. **Flat plate of abdomen** — "survey film of the abdomen reveals a homogeneous density in the left mid-abdomen which extends inferiorly, with its inferior border being identified approximately 3" caudad to the iliac crest. This indicates that the tumor mass which is displacing the stomach as previously described has a fairly sizable inferior component." **Barium enema** — "examination including double contract with air shows that the descending colon is displaced pos-

teriorly by the inferior component of the previously described mass. At the mid-transverse colon, just to the left of the level of the lumbar spine, the caliber is slightly diminished. The lateral projections show that the transverse colon is displayed anteriorly. There is no definite evidence of intrinsic involvement of the colon at this site. **Summary**: the examination of the colon gives additional information as to the nature of this mass, revealing that the descending colon is displaced posteriorly and laterally and the transverse colon is displaced anteriorly. No definite evidence of intrinsic involvement is identified." **I.V.P.**—"shows excellent visualization of both urinary tracts. The kidneys are not displaced. There is a suggestion of very minimal medial displacement of the middle third of the left ureter. This study indicated that the large mass previously described does not have a retroperitoneal origin; although it may have a very slight posterior component capable of displacing the middle third of the left ureter slightly medially. The examination of the urinary channels is otherwise entirely negative". **Small intestines** — "At the 3½ hour the soft tissue outline of the tumor mass is very well delineated. It measures approximately 22 cm. in its longest diameter. It is noted that the jejunal loops are flattened and displaced medially by the tumor mass. Several of the loops are slightly dilated indicating incomplete obstruction by extrinsic pressure. There is no definite evidence of invasion or destruction of the mucosa of the jejunal loops. **Summary**: Large tumor mass, lobulated, measuring approximately 22 cm. in its longest diameter and displacing the distal 2/3 of the stomach medially and posteriorly, the jejunal loops medially, the transverse colon anteriorly and the descending colon posteriorly and laterally. The tumor mass is not retroperitoneal in origin. No intrinsic origin within the gastrointestinal tract has been identified although of course there is evidence of extensive extrinsic pressure For this reason, we discard the possibility of a mucoid carcinoma of the colon with extensive extraluminal growth. It is felt that the large tumor mass represents a large cystic tumor (3 hr. film shows a localized area of displacement suggesting that it is produced by a daughter cyst). The cystic tumor may be of peritoneum origin such as mesenteric or omental cyst or even an enteric cyst".

Laboratory Studies: Serology - negative. Urinalysis: color - yellow clear; reaction - alkaline; Spec. gravity - 1015; albumin - negative; sugar - negative. Feces - Schistosoma mansoni, ascaris lumbricoides. Occult blood - negative. Biopsy of rectal mucosa - positive for Schistosoma mansoni ova; dead, pigmented - 166; dead, collapsed - 30; living ova - one. Cephalin flocculation: 24 hr. negative; 48 hr. negative. Gastric analysis: total acidity - 36 free

Hcl. 28. Blood chemistry: total protein GB& - 7.4%; albumin - 5.08%; globulin - 2.32%. Serum lipase - 0.12 cc of N/20 NaOH. Serum amylase - 166 mod. Somogyi units. Prothrombin: patient: 14 sec. control: 11 sec. Bleeding time - 2 min.; coagulation time - 5'10". W.B.C. - 13,200; Differential count - neutrophils 62; lymphocytes 8; monocytes 8; eosinophils 4; basophils 1; R.B.C. - 3,730,000; hemoglobin 75% coagulation time 10.9 gms; hematocrit 40 mm; juveniles 2%; stabs 3%; intermediate lymphs 12%; RBC appears to be hypochromic. Sedimentation rate - 1 hr. 38 mm. Amylase - 305. mod. Somogyi units; hematocrit 52 mm. Icterus index - 19 units. Blood culture - no growth after 72 hours. NPN 27.7 mgm.%; CO_2Vol.% 36 vol.%; serum chlorides 270 mgm.%; total proteins GM % 7.0; albumin 4.5; globulin 2.5; CO_2ME 16 ME/L.

A WBC of 13,000 with normal differential and RBC of 3.7 m. with 75% hgb; the stools showed 2 to 4 alive schistosoma ova and some ascaris ova. The rectal biopsy contained about 200 dead ova. Sigmoidoscopy to 5" revealed atrophic mucosa. X-rays of the chest were negative. The patient had liver function tests which were essentially negative including BSP and serum proteins, and he also had an amylase of 166 units and a normal serum lipase. X-ray studies of the gastrointestinal tract will be described by the radiologist.

DISCUSSION BY MEMBERS OF THE STAFF

Dr. V: Study of the upper GI tract reveals definite evidence of an extrinsic pressure at the distal 2/3 of the stomach, displacing this organ medially and posteriorly. The stomach is displaced by a large mass. This is well demonstrated on the flat film of the abdomen showing that the mass extends inferiorly 3" below the left iliac crest. The I.V.P. shows no abnormality; this indicated that the mass does not have a retroperitoneal origin; there is a questionable medial displacement of the middle 1/2 of the left ureter. The barium enema shows posterior displacement of the descending colon and also somewhat laterally; the transverse colon is displaced anteriorly; there is no evidence of an intrinsic involvement. Small bowel study shows on the 3 1/2 hr. film the mass well delineated. It measures 22 cm. in its longest diameter. The jejunal loops are flattened and displaced medially by the tumor mass; several of the loops are slightly dilated indicating incomplete obstruction by extrinsic pressure. There is no definite evidence of invasion or destruction of the mucosa of the jejunal loops. In summary, I believe that we have here a large tumor mass, lobulated, approximately 22 cm. in its longest diameter, displacing the distal 2/3 of the

stomach medially and posteriorly, the jejunal loops medially, the transverse colon anteriorly and the descending colon posteriorly and laterally. The tumor mass is not retroperitoneal in origin. No intrinsic origin within the GI tract has been identified although, of course, there is evidence of extensive extrinsic pressure. For this reason, I discard the possibility of a mucoid carcinoma of the colon with extensive extraluminal growth. The tumor may represent a large cyst, perhaps with some daughter cyst, such as mesenteric, or omental or even an enteric cyst. This is what I have reported originally following the respective x-ray examinations. Reviewing the films, I feel that this is a rather unusual type of lesion and the possibility of an argentaffinoma and lymphosarcoma has to be included in the differential diagnosis.

Dr. R. M.: There is one other possibility that I believe can be considered in this case: a schistosomiasis granuloma or rather a granulomatous lesion produced by Schistosoma mansoni. We must not forget that this man has had schistosomiasis for a number of years and that he comes from Utuado where infestation is more likely to occur early in life. I have questioned him quite carefully as to symptoms that could be ascribed to Schistosoma mansoni infection; however, he offers none. I have seen fairly large masses at operation (splenectomy) particularly in the pelvis, consisting of schistosoma granulomatous tissue, mostly fibrous tissue. There are several points, however, against this possibility in this case: First, the man does not present symptoms or signs of liver dysfunction which has occurred in my experience in cases of schistosomiasis presenting a similar mass. And second, the patient has no apparent hepatosplenomegaly.

Dr. L: We referred the case twice to the consulting gastroenterologist and this is his first report: "according to the patient GI symptoms always are preceded by the onset of marked and prolonged constipation. Abdominal examination unsatisfactory. Suggest upper GI series, barium enema and sigmoidoscopy". The second time he was referred, this is his report: "examination of abdomen today reveals a definite hard fullness in the upper epigastrium and left upper abdomen which has no definite borders. There is slight movement of this mass with respiration, it can be pushed from behind and felt anteriorly and vice versa. Suggest glucose tolerance curve, amylase and lipase studies and abdominal exploration".

Dr. E: I somewhat disagree with the x-ray interpretation for the following reasons: I find it peculiar that on the early films of the small bowel study the large abdominal mass on the left side does not displace the jejunal segments; on the 3½ hr. film, some

of the ileal segments are definitely abnormal. This suggests to me that there is intrinsic abnormality of these ileal segments. In addition, I feel that the transverse colon is also involved. Whether we are dealing with an inflammatory or a neoplastic condition, I am not prepared to say. If I am allowed to guess, I favor a malignancy of the transverse colon with perforation in order to explain the large mass and the involvement of the ileal segments.

Dr. A: I would like to explain everything including the original serious illness and acute episode of pain on one diagnosis. I am inclined towards the diagnosis of an acute pancreatitis of the tail of the pancreas with the residual of either a chronic cystic mass or diffuse inflammatory process of the mesocolon, of the transverse colon and descending colon.

Dr. P: This is a very difficult type of tumor to diagnose as are all these free abdominal tumors. It is specially difficult in trying to correlate the x-ray findings, the physical findings and the history of the case. From the physical examination point of view it gives the impression to me that the tumor is an anterior tumor. I felt that it was attached to the anterior abdominal wall but now considering the pushing of the transverse colon forward by the tumor which presupposes a tumor behind the transverse colon, then the picture varies. Were it not for this fact, I would think that the tumor was attached to the anterior abdominal wall. If the tumor is supposed to have developed after the acute episode of illness that the patient had a month ago, then we have to explain the present situation from that point of view, although the presence of the tumor might have been there before the acute attack that the patient had a month ago. This may be an entirely independent affair. If we are going to consider that the present condition is a result of what happened a month ago when the man went into collapse and had some sort of peritonitis, although there is no history of fever, then we have to think that it has been either a hemorrhage or an acute pancreatitis. Some cysts of the pancreas develop in such strange positions; anything might be a cyst of the pancreas; it certainly is rather unusual in the position that this tumor is to call it a cyst of the pancreas. I would be more inclined to think of a cyst of the mesentery into which a hemorrhage has developed, making it grow fast. That is also difficult to consider, difficult to tell also the amount of invasion that that tumor has in the small intestine and large intestine although according to Dr. E., there is a suggestion of infiltration, specially in the jejunal and iliac parts of the small bowel. A lymphosarcoma, is a strong possibility. On the physical examination of the patient the tumor is felt anteriorly when the abdomen is flexed, it can be

pushed posteriorly, and it can be felt also in the costovertebral angles. It does not move with respiration, but it does not have the ample movement that other organs have, like the spleen. As I said before, were it not for the posibility of tumor of the large bowel, of the transverse colon being pushed forward this actually presupposes that the tumor is posterior to the omentum. I think of some tumor attached to the anterior abdominal wall. I do not know if it is possible for such a tumor to press the large bowel, specially the descending colon so much backwards as to show that much deformity. It might be, but it is difficult to conceive also. I was thinking of my past experience with desmoid tumors of the abdominal wall or hemorrhage into the abdominal wall, localized in the form of tumors, but never saw in the x-rays, because no x-rays were taken in these particular patients. I think actually it is difficult to commit myself in anything in this particular tumor although in the consultation I put down a hemorrhage in the abdominal wall.

Dr. E.: Dr. R. mentions the possibility of a perforation due to ascaris because the patient is full with ascaris.

Summary of Preoperative Diagnoses: Dr. A.L.: Residual of pancreatitis; Dr. V: Mucoid CA of transverse colon; Dr. E.: Tumor arising from the transverse colon with perforation involving surrounding structures; Dr. L: Ruptured pancreatic cyst; Dr. S: Pancreatic pseudo-cyst. (Same as Dr. A.); Dr. R. Pancreatic cyst. Ascaris lumbricoides - perforation with abscess; Dr. M: Lymphoma; Dr. R.: Lymphosarcoma; Dr. G. P.: Lymphosarcoma; Dr. R.M.: Schistosoma granuloma; Dr. P.: Tumor, anterior abdominal wall.

Operation Report: The operation was performed on April 22, 1954. **Findings:** 1. A large cystic mass with well defined walls and considerable pericystic inflammatory reaction, measuring approximately 6" in length and 3" in width, was found attached along the left rectus muscle of the anterior abdominal wall and slightly toward the lateral abdominal wall. The walls of the cyst were found adherent to several coils of the small intestine and to the descending colon. The attachment to the abdominal wall could not be separated by blunt dissection and it included all of the anterior surface of the cyst, so that the main and most extensive portion of the cyst was attached to the abdominal wall, and the cyst, from there, extended into the peritoneal cavity in the form of a hemisphere. The attachment to the small bowel was in several small areas of not more than 1 or 2 cm. in size. The attachment to the anterior wall of the colon was in 3 areas, two small and the third nearly 1½" long and 3 4" wide; this one was more intimately attached and after separation of the mass, the wall of the descend-

ing colon presented a small area of superficial granulation that could be excised well, and the wall proper was somewhat indurated, representing an area of localized colitis. There was no narrowing of the lumen of the colon and the mesenteric and lateral aspects of the colon were perfectly normal. The attachment of the cyst to the colon was considered minimal compared to the attachment of the cyst to the adbominal wall. The cyst cavity contained about 300 cc of creamy yellowish liquid. 2—The rest of the descending colon and sigmoid was thoroughly examined and no abnormality was found. 3—The stomach appeared entirely normal. 4—On the first portion of the duodenum there was a small area of scarring consistent with a previous duodenal ulcer. 5—The gall bladder was entirely negative. The pancreas appeared entirely normal to palpation. The liver substance was normal in color and consistency. **Operation:** 1—Under endotracheal gas, oxygen, ether anesthesia and after the usual preparation and draping the peritoneal cavity was entered thru a left rectus splitting incision which extended from the left lower quadrant to the region of the xyphoid. 2—The abdominal cavity was explored with findings as listed above. 3—The cyst cavity was separated from all the adherent structures with sharp and blunt dissection. A stab wound was made in the left upper quadrant and a Penrose drain was introduced into the abscess extraperitoneally at the first portion of the operation. 4—A culture was obtained from the contents of the cyst and dissection of the mass was continued when it became evident that complete removal could be performed. 5—The wall of the cyst was grasped and placed on traction and further off from the bowel, anterior adbominal wall, left lobe of the liver was performed until the abscess was removed. 6—Three Penrose drains were led out thru a stab wound incision on the left side of the abdominal wall. 7—The operative area was irrigated with saline solution and closure of the incision was performed with continuous #1 chromic catgut to peritoneum and posterior rectus sheath and interrupted #0 chromic catgut to the anterior rectus sheath. Interrupted fine silk was used for the skin. 8—Patient received 1½ blood transfusions while on the operating table and withstood the procedure well except for some anoxia at the early part of the procedure.

Comments: The cyst was full with yellowish, milky content, nearly 300 cc, with definite walls, give the impression of a dermoid cyst. Nevertheless, to explain the clinical picture, it could be thought that there has been a perforation of the bowel with localized peritonitis and abscess formation, although the content of the cyst was absolutely odorless.

Pathological Examination: Gross Description — the surgical material includes three separated specimens: (A) It consists of a fibrous cystic or abscess capsule that measured 6 cm. in diameter and several hemorrhagic inflammatory omental segments. The wall of the fibrous capsule is fibrotic, grayish-pink, and its inner surface is corrugated and covered with a fibrinous exudate. In addition, there are several small segments of tissue pertaining to the capsule. Representative sections are taken from the capsule and omental tissue for histologic examination. (B) It includes three pieces of membranous tissue similar to that of the fibrous capsule of specimen A. (C) It consists of a small, soft lymph node 1 cm. in diameter. Its sectioned surfaces are grayish-pink and homogenous. All embedded. **Histologic examination:** The wall of the abscess structure consists of dense collagenized fibrous connective tissue. The capsule is well vascularized and it presents a granulomatous inflammatory reaction manifested by a perivascular infiltrate of plasma cells, lymphocytes, and eosinophils. The inner surface of the abscess wall is lined with fibrinous material and tissue debris, resting upon a narrow inflammatory zone including round cells and histiocytic macrophages. There is a differentiated or specific granuloma in one sector corresponding to the outer portion of the capsule. This granulomatous area is made up of Schistosoma mansoni pseudotubercles including pigmented eggs with dead miracidia lying toward the center of the epithelioid nodules. The stroma between the nodules is closely infiltrated with lymphocytes, plasma cells, and eosinophils. Dead collapsed and pigmented Schistosoma mansoni eggs are found in the fibrous wall of the abscess. The segment of omentum presents a non-specific chronic inflammatory process and hemorrhages. The lymph node shows evidence of reactive hyperplasia. **Diagnoses: (1) Schistosoma mansoni granuloma with abscess formation, paracolic:** adherent to the ascending colon and anterior abdominal wall. (2) Reactive hyperplasia of **lymph node, mesenteric.**

CARCINOMA OF THE STOMACH

LUIS A. PASSALACQUA, M.D., and HECTOR M. NADAL, M.D.***

The present report comprises a series of 71 patients in which a positive diagnosis of malignancy of the stomach was made by the study of pathological specimens.

Type of lesion

One of the patients had two simultaneous cancers, consisting of an adenocarcinoma of the stomach and one squamous cell carcinoma of the middle third of the esophagus. A diagnosis of lymphosarcoma was made in one of the cases and of leiomyosarcoma in another. The remaining 68 patients had adenocarcinomas.

Operative procedures

TABLE 1

Number of patients studied	71
Treatment:	
No operation	2
Laparotomy and biopsy	7
Palliative (Non-resection)	8
Gastrectomy, subtotal	33
Gastrectomy, total	21

In two patients the lesion was so far advanced and the condition of the patient so poor that no operative procedure was performed outside of a needle biopsy of the liver in one and removal of a cervical lymph node on the other. The tumor was considered not resectable after laparotomy in 7 patients, or 9½ per cent of all cases. A section of the tumor was removed in all of these cases for pathological study. No palliative surgery was performed in any of them.

Eight of the patients had palliative surgery consisting of gastrostomy, gastroenterostomy or side tracking esophagojejunostomy.

---

* Chief, Surgical Service, VA Hospital, San Juan, Puerto Rico.

** Surgeon, VA Hospital, San Juan, Puerto Rico.

*** This statistical study extends from January 1, 1947 to November 30, 1955.

NOTE: The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

The number of patients in which resection was performed was 54, as follows: total gastrectomy plus esophagectomy 1; total gastrectomy including partial pancreatectomy and splenectomy 10; total gastrectomy 10; subtotal gastrectomy with removal of omentum 33.

Immediate mortality

Among the group in which resection of the tumor was made there were 4 deaths, a mortality of 7.4 per cent. One death occurred on the 10th day after the operation of suppurative mediastinitis and empyema following sloughing and separation of the line of sutures of the esophago-duodenostomy.

In the total gastrectomy group there were 2 deaths, a mortality of 9.5 per cent. One death was due to atelectasis and pneumonitis on the fourth postoperative day. The second death was due to peritonitis following dehiscence of the wound. In both patients the operations were performed through a thoracoabdominal incision.

Only one death occurred among the 33 patients in which a subtotal gastrectomy with removal of the omentum was done, a mortality of 3 per cent. This death occurred on the third postoperative day on a patient 73 years old who developed atelectasis and pneumonitis.

Operative complications

TABLE 2

Wound dehiscence	5
Wound sepsis	5
Bronchopneumonia	2
Transient diarrhea	2
Transient diabetes	2
Phlebothrombosis	2
Jejuno-colic fistula	1
Esophago-pleural fistula	1
Partial obstruction large bowel	1
Persistent fever	1
Hepatitis	1

There were 23 complications recorded in 18 patients. (Table 2) Incomplete wound dehiscence occurred in 5 patients. One of these patients died as explained before. Wound infection or hematoma developed in 5 cases. Pneumonia following atelectasis developed in two patients and both patients died. Two patients developed diarrhea which was not serious and recovery was established in a

few days. Among the patients in which partial pancreatectomy was performed during the total gastrectomy, 2 developed transient diabetes. Phlebothrombosis developed in 2 patients with recovery, after being placed on anticoagulant therapy. One patient had a jejunocolic fistula which was reoperated and he lived four years after the operation. The fistulous tract was examined pathologically and found to contain carcinoma. There was one esophagopleural fistula and one patient developed obstruction of the large intestine.

Resectability

TABLE 3

Resectable	54
Non-resectable	17
Total	71
Percentage resected	76%

Of the 71 cases, 54 were resectable and 17 were not resectable. The percentage of resectability was 76 per cent. (Table 3)

Age

The average age was 61 years. The youngest patient was 26 years old and the oldest 76 years of age.

Presenting symptoms

In 63 per cent of the patients the presenting symptom was epigastric pain, while in 20 per cent the only complaint was epigastric discomfort. Weakness constituted an initial complaint in 5 per cent of the patients.

Survival time of all resected cases

TABLE 4

No. Yrs.	No. Pts.	Dead	Alive
0-1	30	25	5
1-2	7	4	3
2-3	3	2	1
3-4	7	1	6
4-5	2	2	0
5-6	3	0	3
6-7	1	0	1
7-8	1	0	1
TOTAL	54	34	20

Table 4 represents in tabulated form, a survival summary of all resected cases.

Survival following Palliative Surgery

TABLE 5

1 patient	1 month
2 patients	2 months
1 patient	6 months
1 patient	7 months
2 patients	8 months
1 patient	5 years 5 months*

* No biopsy first surgical procedure — 1948.

In the group undergoing palliative surgery (Table 5) one patient lived one month, two patients lived 2 months, one patient 6 months, one patient 7 months, two patients 8 months, making an average survival of 4 months and 24 days.

One patient had a gastroenterostomy performed on February 24, 1948 for what appeared to be grossly an unresectable carcinoma of the stomach. There was no glandular enlargement and no metastasis present. The tumor was adherent to the liver. This patient died five years and five months later of generalized carcinomatosis. Since no biopsy was made the first time, we do not feel absolutely sure of the previous diagnosis, specially after having such a prolonged survival.

Survival of the unresectable patients

TABLE 6

A. Laparotomy and Biopsy

1 patient	1 month
1 patient	3 months
4 patients	3 months
1 patient	4 months

B. Cervical biopsy only (No exploration)

2 patients	1 month

The 7 patients that had only exploratory laparotomy and biopsy lived an average of forty six days. (Table 6)

Two patients that were not explored lived an average of 28 days. One of the patients was a 66 year old male who came to the hospital with a complaint of generalized adenopathy. He was found to have on admission multiple subcutaneous nodules in the cervical region, the axilla and the inguinal regions. A biopsy of the cervical lymph nodes revealed metastatic carcinoma. A postmortem

was performed one month after admission revealing an anaplastic carcinoma of the stomach with local extension to the duodenum, but there were no metastases in the regional lymph nodes, in the intraperitoneal viscera including the liver or in the lungs. Nevertheless, there was a large metastatic node in the left auricle of the heart and the lymph nodes of the neck, axiliary and inguinal regions.

Survival of patients with total gastrectomy

TABLE 7

0-1	year	2 pts.
1-2	years	2 "
2-3	"	1 "
3-4	"	1 "
4-5	"	0 "
5-6	"	1 "

There were 20 patients who underwent total gastrectomy, two of which included partial pancreatectomy and splenectomy. (Table 7). Of these, 7 patients are alive today, a survival of 35 per cent. Of these, 2 patients who had a total gastrectomy are alive less than one year. Two patients who had total gastrectomy plus partial pancreatectomy and splenectomy are living between one and two years. One patient who had a partial pancreatectomy, splenectomy and total gastrectomy is living between two and three years. One patient who had a total gastrectomy, partial pancreatectomy and splenectomy is living between three and four years. One patient who had a total gastrectomy only is living now six years. The average survival of the patients living today is 2 years and 5 months.

Total gastrectomy - Over all survival

TABLE 8

No. Yrs.	No. Pts.	Dead	Alive
0-1	13	11	2
1-2	4	2	2
2-3	1	0	1
3-4	1	0	1
4-5	0	0	0
5-6	1	0	1

Table 8 illustrates the over all survival of the total gastrectomized cases.

Survival of patients having subtotal gastrectomy

TABLE 9

0-1 year		3 pts.
1-2 "		1 "
2-3 "		0 "
3-4 "		5 "
4-5 "		0 "
5-6 "		2 "
6-7 "		1 "
7-8 "		1 "

The subtotal gastrectomy group comprises 33 patients. At present there are 13 patients living making a total 39-1 3 per cent (Table 9). Of these, there are 3 patients living less than one year after the resection. One patient is living between one and two years, 5 patients are living between three and four years and 2 patients between five and six years. There is one patient who is alive after six years and one patient over seven years.

TABLE 10

No. Yrs.	No. Pts.	Dead	Alive
0-1	16	13	3
1-2	3	2	1
2-3	2	2	0
3-4	6	1	5
4-5	2	2	0
5-6	2	0	2
6-7	1	0	1
7-8	1	0	1

Table 10 illustrates overall survival time of those patients undergoing subtotal gastrectomy with removal of omentum.

Carcinoma of the Stomach - Over 5 years survival

TABLE 11

Number of cases operated up to 1950	20
Alive today	5
Per cent survival 5 yrs. or over	25%

There were 20 cases operated up to 1950. Of these, there are 5 alive today, giving therefore a 25% survival of five years or over. (Table 11).

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º Part Time
H or RO followed by *, spends some time at Hospital and/or Regional Office.

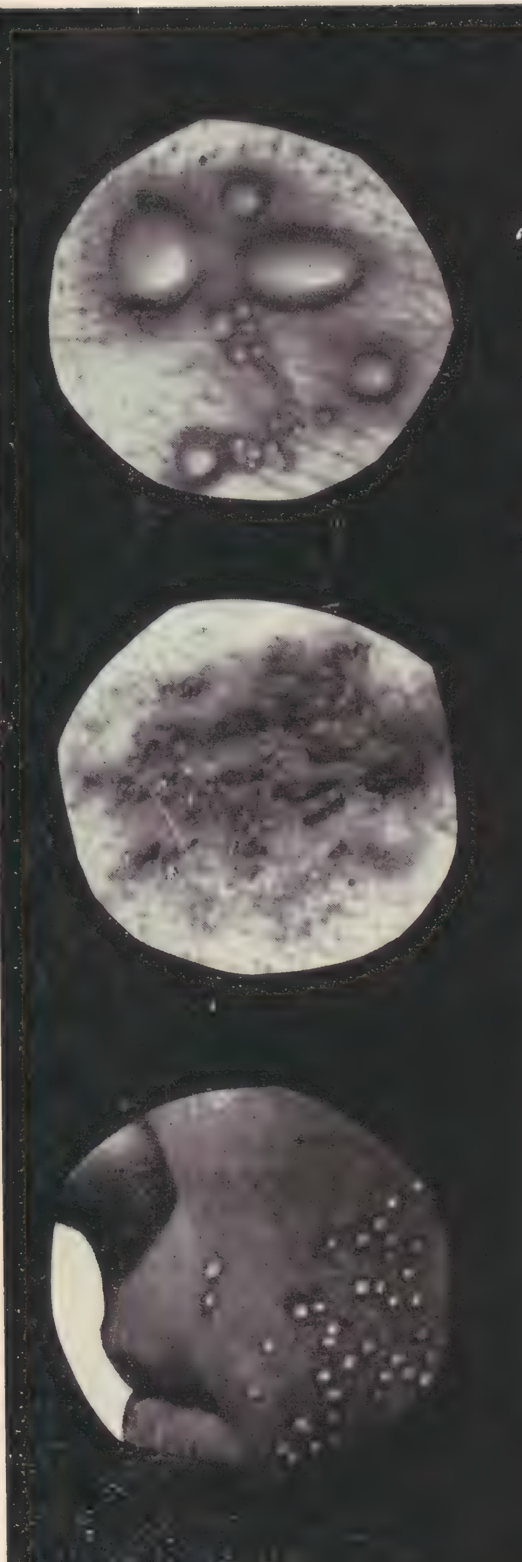
SUMMARY

	Doctors	Dentists	Non-Medical
Full Time	27	4	0
Part Time	14	0	0
Consultants	15	3	3
Attendings	7	–	–
Residents, Medicine	3	–	–
Residents, Surgery	3	–	–
Residents, Radiology	0	–	–
TOTAL	69	7	3 = 79

	Hospital	Regional Office
Full Time Doctors	15	12
Part Time Doctors	8	6
Full Time Dentists	4	0
Attendings	3	4
Consultants, Medical	14	1
Consultants, Non-Medical	3	0
Consultants, Dental	3	0
Residents	6	0
TOTAL	56	23 = 79



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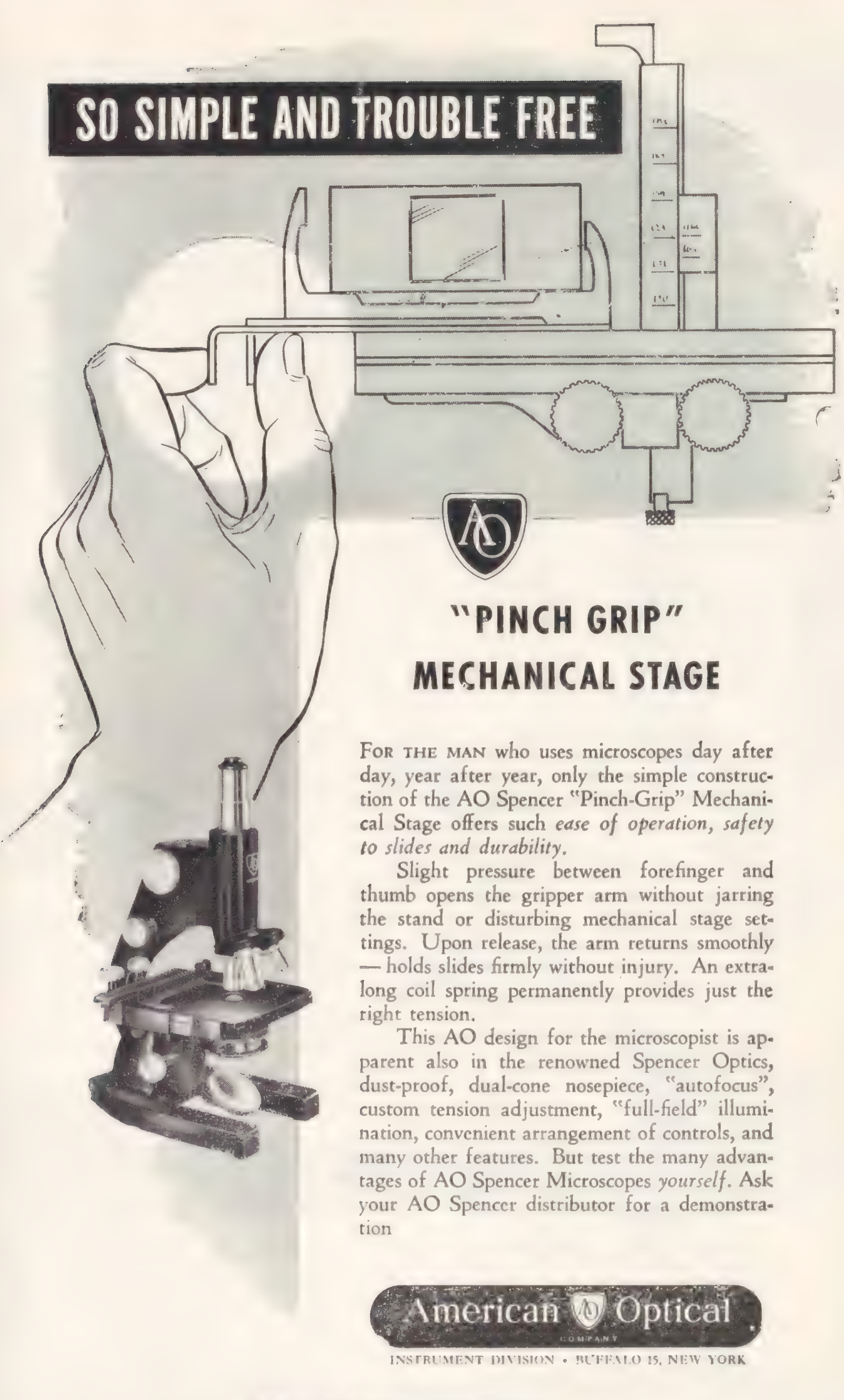
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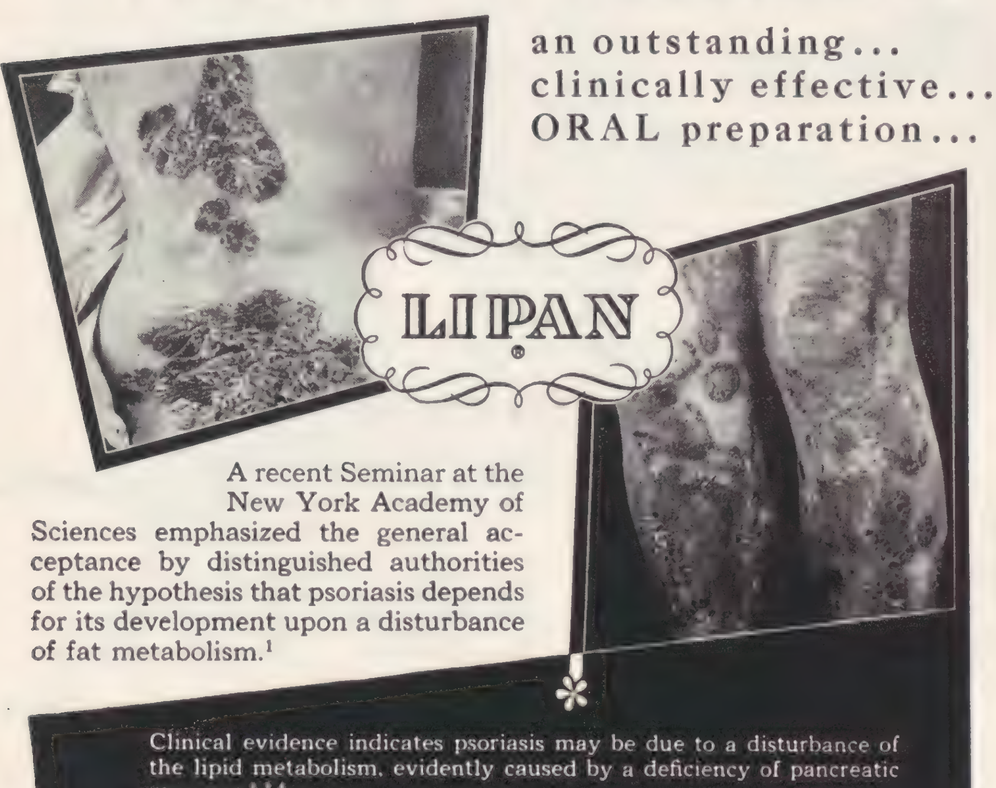
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4. Combes, F. C., Management of Psoriasis As a Metabolic Lipid Disturbance. New York State Journal Medicine, 54:13 (July 1954).

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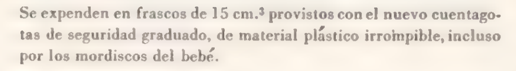
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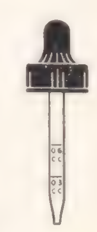
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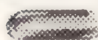
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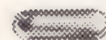

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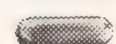
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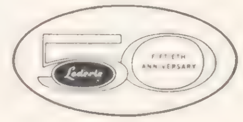
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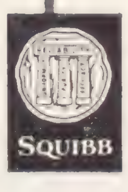
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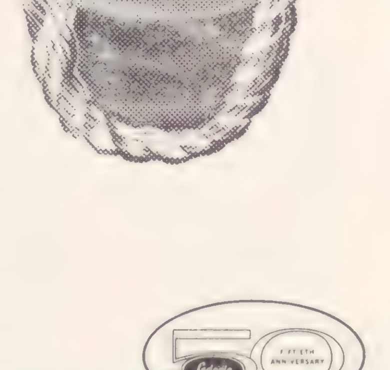
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 48 SEPTIEMBRE, 1956 No. 9

CHYLURIA

*B. RODRIGUEZ-LUCCA**

The term chyluria is applied to the passage of chyle in the urine. The color of the urine varies with the amount of chyle it contains. The urine may be opalescent, milky, creamy, or may contain translucent clots. It contains fat, white and red blood cells, albumin, globulin, and fibrinogen. When both chyle and blood are present in considerable amounts, the term hematochyluria is applied.

Chyluria is caused by a ruptured lymphatic varix which communicates with the urinary tract. The varix is usually due to retroperitoneal lymphatic obstruction. The ruptured lymph varix may empty into the kidney, ureter, bladder, or posterior urethra, but usually occurs in the upper urinary tract. The most common cause of retroperitoneal lymphatic obstruction in Puerto Rico is Filariasis.

The filarial worm, Wuchereria bancrofti, is a thin nematode about one half centimeter in length. Their larvae also known as microfilaria, is transmitted by the bite of the Culex, Aedes, and Anopheles mosquitoes. They penetrate the human skin by the insect's bite, then follow the superficial lymphatics to the deep lymphatic vessels and nodes where they lodge. The lymphatics of the retroperitoneal space are most frequently involved and there the microfilaria become adult filarial worms. The presence of a filarial worm within a lymph node or vessel produces an inflammatory reaction with many eosinophils, plasma cells, giant cells, and polymorphonuclear leukocytes. This is followed by fibrosis and results in the occlusion of the lymphatic, leading to the formation of a lymphatic varix.

Chyluria is an alarming symptom to the patient who promptly comes for medical advise because he is passing milky urine. Sometimes the patient has difficulties in emptying his bladder

* From the department of Surgery, Section on Urology, of the San Juan City Hospital and the University of Puerto Rico Medical School.

because of the formation of chylous clots. This is more so on patients who have residual urine because of vesical neck obstruction. The classical symptoms of chills and fevers of the acute phase of Filariasis are usually absent since chyluria is a late manifestation of the disease.

The diagnosis of chyluria can be made from gross examination of the urine. It is best confirmed by extracting the chyle with a fat solvent such as ether. After mixing equal parts of ether and urine in a test tube, the chylous urine becomes clear. The fat globules can be stained with Suddan III or any other fat stain.

The site of the ruptured varix may be visualized cystoscopically if it is in the bladder, vesical neck, or posterior urethra. However, most frequently the ruptured varix is in the upper urinary tract. Chylous urine can be readily seen spurting from one or both ureteral orifices. By gradually withdrawing a ureteral catheter from the renal pelvis the site of the ruptured varix can be ascertained. If the varix is in the ureter, clear urine will be obtained from the renal pelvis. If the varix is in the renal pelvis, the urine will be chylous all the time. When the tip of the ureteral catheter reaches the level of the ruptured lymphatic varix the clear urine becomes chylous. In some instances, the varix may be demonstrated radiographically by ascending pyelography.

The treatment of chyluria consists mostly of reassurance to the alarmed patient who seeks medical advise because he is passing milky urine. In some cases the patient looses considerable fat and protein in the urine and may have symptoms of weakness and weight loss. Again the treatment is conservative with dietary measures and rest. Renal decapsulation and nephrectomy are mentioned only to be condemned.

If the patient has filariasis, Hetrazan, brand of 1-diethyl carbamyl-4-methylpiperazine hydrochloride in doses of 2 milligrams per kilo three times a day for three weeks is the treatment of choice. This will cure the filariasis but not the chyluria which is the result of a dead adult worm obstructing a lymphatic channel.

During the past year I studied seven new patients with chyluria in the Urology Clinic of the San Juan City Hospital. There were two males and five females. The youngest patient was a nine year old girl and the oldest one was sixty-seven. The site of the ruptured varix was not visualized in any of them since the chylous urine was coming from the upper urinary tract. There were three patients with positive blood smears for microfilaria and in one of these microfilaria were also identified in the urine. There was eosinophilia in five cases and in the remaining two cases was in the upper limits of normal.

CHYLURIA* - Analysis of Seven Cases

Name	*Age*	*Sex*	*Clots*	*Origin*	*Pyelogram*	*Filarial Blood*	*Smears Urine*	*Eosino-philia*
E. G.	16	F	++	Right kidney	Normal	—	—	13%
L. R.	52	M	—	Right ureter	Normal	—	—	14%
V. R.	67	M	++++	Left kidney	Normal	—	—	3%
M. J.	9	F	+	Right kidney	Normal	—	—	35%
C. V.	19	F	—	Left ureter	Normal	+	+	8%
C. B.	55	F	—	Right kidney	Normal	+	—	4%
G. M.	22	F	—	Right kidney	Normal	+	—	14%

SUMMARY AND CONCLUSIONS

Chyluria is a urologic condition not infrequently encountered in tropical and subtropical areas where Filariasis is endemic.

The treatment of the condition is conservative.

Seven cases of chyluria seen during the past year at the San Juan City Hospital are presented.

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1602 Loíza Street
Santurce, Puerto Rico.

GENERAL CONSIDERATIONS ON THE MANAGEMENT OF ASTHMA IN CHILDREN

JOSEPH L. APONTE, M.D.

Asthma is not the most common manifestation of allergy but it is the most important one. It is a chronic, disabling and incapaciting disease, both to the parent and child. Many a child grows permanently handicapped physically and emotionally, a cripple and a nervous wreck, because of a chronic asthmatic condition. The management of a child with chronic asthma is a challenge to the family physician, who usually has from the beginning a pessimistic attitude and a feeling of defeat. This is readily transmitted to patient and parents.

A child is considered as suffering from chronic bronchial asthma if he has attacks through the year which have persisted despite all efforts of treatment. This recurrent attacks are characterized by a prodromal stage of sneezing, runny nose, itchy eyes, persistent dry cough, headache and sometimes fever. This prodromal stage last from a few hours to 3-4 days. Its recognition is very important for if the treatment is started early the asthmatic attack can be aborted. Then comes the asthmatic attack: It is a form of obstructive emphysema, characterized by expiratory dyspnea, of bilateral involvement (generalized), manifested by wheezing, usually fine and bilateral and due to a combination of three factors: smooth muscle spasm, edema of mucous membranes, and accumulation of secretions in bronchi with plugging. With such definition in mind the therapeutic approach is simplified.[1]

Recurrent paroxysmal attacks of dyspnea, chiefly expiratory, and with bilateral generalized wheezing, in a patient with a family history of allergy and or a personal history of previous allergic manifestations, is asthma until proven otherwise. This picture will rarely be reproduced by some other conditions, but certain syndromes can cause difficulty in differential diagnosis because they may fulfill one or more criteria of the above definition.

Acute respiratory difficulties of obstructive nature often cause difficulty. Laryngeal and tracheal lesions such as congenital laryngeal stridor, croup, laryngotracheobronchitis, diphtheria, and foreign body in trachea sometimes simulate an asthmatic attack. But they usually show hoarseness, aphonia and brassy cough, plus the fact that the retraction is inspiratory and not expiratory like in asthma. Other less common obstructive lesions are enlarged tuberculous bronchial or hilar nodes, mediastinal masses such as

Hodgkin's disease and lymphosarcoma, substernal thyroid, retropharyngeal abscess, tracheal stenosis, rings or papillomas, pneumothorax.

Non obstructive lesions giving acute obstructive symptoms are: bronchopneumonia, other infections such as pertussis, bronchiectasis, and lung abcess. Irritation of the lungs from dust fumes and gases, cystic fibrosis of the pancreas, some types of congenital heart disease and heart failure from heart or renal disease.

Prophylaxis:

There has been increasing interest in the last few years on the application of prophylactic measures to the allergy problem in children. Allergic diseases have hereditary or constitutional factors involved. So that if one or both parents suffer from some major allergic disease, the chances are that the offsprings may be affected sometimes during their lifetime, but usually during childhood. Thus a potentially allergic child is one that has at least one allergic parent or sibling. It is also postulated that a physiological immunological immaturity exists in the early months of life that results in sensitization to unaltered food proteins. If cows milk proteins which constitutes the chief source of food and is the commonest form of food allergy in the first year of life, were completely withheld from the infants diet until this period of physiological immunological immaturity passed, the allergic symptoms might be minimized or prevented. When cows milk is then added to the diet it is usually well tolerated. Babies can be succesfully raised on a milk free diet since birth. Soy bean milk and meat base milk are adequate substitutes. Of course if breast milk is used instead cows milk, the whole problem is obviated.

With this considerations in mind, Dr. Jerome Glaser[2] and his group from Rochester, N. Y., conducted a controlled study. Ninety six potentially allergic children were started on a milk free diet from the moment of birth, 88 were given soy bean milk, 3 were breast fed and five were given meat base formulas. In order to minimize the chances of sensitization through breast, milk, cheese and cheese products were limited on the mother's diet.

The above infants were exposed to cows milk after 6-9 months of age. They were followed for 1-10 years. A sibling control group of 65 children and a nonrelated control group of 175 children was used. The results of this study showed that the incidence of major allergy was 15% in the experimental group compared with 64% of the sibling group and 52% of the nonrelated control

group. The authors concluded, that in the potentially allergic infant, allergic disease can be prevented in many cases by substituting soy-bean milk or meat base milk for cows milk, withholding cows milk until approximately six months of age, by which time the period of physiologic immunologic immaturity has largely passed.

Other more simple prophylactic measures should be instituted, given a potentially allergic child. Avoid too early addition of other food of high allergenic index such as orange juice, eggs, tomatoes, wheat, corn, nuts, and seafood. It is also very important to recognize and (institute antiallergic measures) treat certain clinical conditions that are precursors of asthma: The idea is to establish the fact that you have an allergic child and treat him as such. Watch for persistent baby colic, urticaria, atopic dermatitis and syndrome known as RURI (recurrent upper respiratory infections). This is the child that always has a cold after the other, this cold is not contagious, has slight constitutional symptoms, itching and rubbing of nose is very common.

Management of the asthmatic patient:

In an excellent discussion on the general management of allergic children, Dr. Bret Ratner[3] states that the adequate and comprehensive treatment of the asthmatic child should involve the following:

1—An approach on the allergic basis, i. e. the extensive and searching investigation through the history, skin testing, elimination of the offending substances, and the building up of an adequate state of immunity over a period of years.

2—A complete investigation of the child's constitutional make up, i. e. diagnosis and treatment of possible anemia, nutritional deficiencies, parasitosis, and hypothyroidism.

3—Constant diagnosis and treatment of the infectious episodes that occur frequently in respiratory allergy in childhood and tend to simulate true asthmatic attacks.

4—Understanding and treatment of the emotional levels involving both the child and his parents.

Environmental control:

The home must be kept as free of house dust as possible. Details instructions should be given. Air conditioning is of great benefit. The child's room should be free of all wool and wool fabrics, feather and kapok products. The pillows and mattresses should be of sponge rubber or glass fiber and covered with dust

proof covers, to prevent dust from settling into the fabric. There should be no animal pets with fur or feathers in the household of an allergic individual, regardless of the results of skin tests. Sensitivity to epidermoids is not easily acquired and the presence of such animals is an invitation to trouble. The patient must avoid irritating fumes and odors such as cigarette smoke, kerosene, moth balls, cleaning fluids and strong perfumes. Avoid humidity and dampness around the house. It favors growth and multiplication of fungi: they represent an important factor in warm climate. Avoid exposure to cold and sudden changes in temperature. Too much emphasis has been placed on the advisability of a change in environment. It has a temporary effect only. It also has sometimes beneficial psychological implications.

Control of emotional factors:

Given an allergic child with asthma, psychological factors may influence the episodes in a number of ways.[4] Certain circumstances that makes the child anxious and unhappy may intensify the allergic symptoms: an unhappy and disturbed home, difficulty with school work such as reading disability, restricted activity, friction with schoolmates or difficulties with the teacher. A bossy or dominating mother may upset the asthmatic child. The child become aware of the agitation produced by his attacks, and the worry exhibited by the parents. He then finds in his illness a ready way of getting his own way and evading responsibilities. The child emotional tension may be increased by sense of guilt, believing that he might have caused his asthma by not following his mothers directions or not taking his medicine. An asthmatic attack is dramatic and frightening, and the fear of an attack produces emotional tension. If he sees his parents disturbed and worried by the attack, his own anxiety increases.

Office management of the child with asthma:

The essential components of the case study are the history, the physical examination and the special tests pertinent to the condition. The history must be real thorough, with reference to prenatal factors, infant feeding, past personal history of allergies, clinical factor involved in presenting allergic difficulty, response to previous therapy, emotional factors involved and detailed description of a typical attack.

Physical examination must be complete. Inspection of nasal mucous membranes yield valuable information. Latent wheezing may be best heard by having the patient exhale forcefully.

The examination of the nasal or bronchial secretions for eosinophiles is a very simple and useful office procedure. A smear loaded with eosinophiles is diagnostic of allergy. Other diagnostic procedures are X Ray of chest and sinuses, rhinolaryngologic consultation and tuberculin testing.

Skin testing:

Skin testing is an aid in the diagnosis of allergic disease if properly interpreted and evaluated. One should never resort to skin testing as a routine procedure before careful history and physical examination. Such tests must be interpreted in the light of the clinical picture, introduction and elimination of offenders and their subsequent effect in such clinical symptoms. It is a comparatively simple procedure. Such tests will frequently give a clue to the type of sensitivity present and in many instances indicate the offending allergen.

Desensitization should be carried out when the offending factors cannot be removed from the patient environment. The injection of small amounts of antigens at regular intervals will built up the patient resistance to his allergens. Desensitization to dust, fungus and pollen allergens is usually a rewarding procedure.

Management of the asthmatic attack:

Early diagnosis and intensive all out therapy is essential. A severe asthmatic attack can be prevented or minimized if proper treatment is instituted early. Asthma commonly is preceded by 2-3 days by a common cold or a common cold-like clinical pictures of sneezing, runny nose, stuffy nose, itchy eyes and cough. Therefore, start treatment at the very first sign of a cold or impending asthma. Do not wait until the patient is dyspneic and wheezing. The routine advocated is the following:

1—Bed rest in a room kept at an even temperature and prepared as allergen free as possible. Steam inhalations are of value to allay coughing and thin out secretions.

2—The simultaneous administration of the following medications every four hours for the first 24-48 hours or until the danger from an asthmatic attack has passed.

a—An antihistaminic drug.

b—A vasoconstricting nose drop.

c—A cough mixture containing an expectorant, ephedrine, or other similar sympathomimetic agent.

If the above measures fail and the patient develops asthma, start an aminophylline suppository of child size (0.25 gm.). Chil-

dren under 3 years old should receive ½ a suppository or less. It can be repeated every 6-12 hours. Continue the cough mixture and the nose drops as needed. Aqueous epinephrine, 1:1000 hypodermically can be used at this stage. It is still the drug of choice for a severe asthmatic attack. It is much better to give children small amounts frequently rather than large doses at wide intervals. I usually give 0.20 to 0.30 cc every 20 minutes for 2-4 doses. Sus-Phrine 1:200 (Brewer) is a slow acting potent aqueous suspension of epinephrine that can be recommended.

Other useful drugs:

Codeine should be used judiciously in cough prescriptions. Watch for signs of overdosage or idiosyncrasy such as drowsiness, depressed respiratory rate and itching around nose and mouth. So called asthma tablets such as Amodrine (Searle) and Tetral (Maltine Company) are useful in selected cases. They contain a sedative, ephedrine, aminophylline and sometimes an antihistaminic. Bronchodilators by inhalations such as Epinephrine Hcl, 1:100, Isuprel (Winthrop-Stearns) and vaponefrin (Vaponephrine Co.) are useful in cooperative older children. Sedation with chloral hydrate or barbiturates should be used as needed to control apprehension and to give the child sufficient rest. Never use morphine.

Antibiotics and or chemotherapy should be administered early in the attack for associated respiratory infection.

The use of ACTH and or Cortisone should be limited to short terms therapy of three to five days for acute asthmatic episodes when the measures already outlined have not been successful in terminating an attack. One should not wait for status asthmaticus to develop to resort to these measures. I prefer to use Cortisone by mouth 100 to 200 milligrams daily. Intravenous hydrocortisone has been used recently effectively for severe attacks. Treatment with these hormones is never a substitute for thorough allergic study and search for specific etiological factors with subsequent specific avoidance or desensitization.

Status asthmaticus

A severe attack that last over 24 hours and fails to respond to an accepted intensive therapy including adrenalin and aminophylline is considered as status asthmaticus. It is rarely seen in private practice, but very common in service patients. It is real pedriatic emergency that sometimes calls for heroic measures. The disease in children is more difficult to treat than in adults.[6]

The mucosa of the tracheobronchial tree in infants is more redundant, thus the lumen is smaller and resistance to airflow (obstruction) is more of a problem. The cough reflex is not so adequately utilized as in adults. Dehydration occurs more readily because of immature kidneys, increase insensible fluid loss, increase sweating and more febrile reactions. Infection is an important contributory factor. The principle of therapy in status asthmaticus is the releive of bronchial obstruction produced by all three mechanisms of spasm, edema and accumulation of secretions in lumen. Inadequate alveolar ventilation produces an increased physiological dead space, and because of arterial unsaturation, carbon dioxide retention may occur. Intoxication ensues and with it central nervous system depression, slowing of respiration and diminishing state of consciousness.

Outline of therapy for status asthmaticus:

1—Prompt hospitalization.

2—Continuous clinical and laboratory evaluation of patient.

- a. Respiratory rate.
- b. Degree of bronchospasm.
- c. Amount and tenacity of secretions.
- d. Superimposed infections.
- e. Cyanosis.
- f. State of hydration.
- g. Muscular fatigue.
- h. State of consciousness.
- l. Acid-base balance.

3—Reinstitute adequate alveolar ventilation:

- a. Drug: Adrenalin is the drug of choice. Given small repeated amounts as stated above. Aminophylline should be continued intravenously in a dose of 0.006 gm per kilogram of body weight (1/20 gr. per pound). Diluted in glucose containing fluids and given very slowly as a drip.
- b. Sedation: Never use morphine. Keep patient sedated with barbiturates or chloral hydrate.
- c. Hydration: Intravenous fluids should be administered not only to restore fluid loss, but also to assist in thinning out the bronchial secretions frequenly inspissated at this stage of the attack. These children are frequently in ketogenic acidosis due to carbohydrate starvation as a result of either vomiting or low food intake.

d. Oxygen: Administer with caution, use low concentrations. In case of tenacious bronchial secretions use steam in the oxygen tent. Detergents are not necessary and may be dangerous.
e. Antibiotics and chemotherapy: They should be continued as previously outlined. Penicillin is the drug of choice for associated respiratory infection. Penicillin sensitivity is rare in children.

SUMMARY

1—Chronic bronchial asthma is common in pediatric practice. It is not a hopeless condition. The physician should deal with this problem with enthusiasm and institute all out therapy because of the multiplicity of factors involved. Asthma is a curable disease.
2—Prophylaxis of allergic disease is briefly discussed.
3—The management of the acute asthmatic attack and of status asthmaticus in children is discussed.

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THE VALUE OF FLUOROSCOPIC EXAMINATION OF THE LUNGS IN CHILDREN*

JOSE E. SIFONTES, M.D., PRISCILA DIAZ DE GARAU, M.D. and CARLOS N. VICENS, M.D.

Fluoroscopic examination of the lungs in children is an extremely valuable procedure; however, there are very few papers devoted to this subject and very little information is available in regard to its limitations and advantages in the practice of Pediatrics.

The purpose of this communication is to report on a brief study designed to determine the value of fluoroscopy of the lungs in children. No attempt shall be made to include fluoroscopy of the heart in this paper.

Materials and Method:

The technic of fluoroscopy of the chest has been described in texts on diagnostic roentgenology.[1] In this study dark glasses were used for ten minutes before entering the fluoroscopic room; the patients were examined in the upright position and observations made in the dorsoventral, right oblique, left oblique and ventro dorsal positions. The fluoroscope was set at 3 milliampers and 60 kilovolts. The observers were the authors of this paper. Each one of us knew a few of the patients studied, but not all of them; the patients were selected among children, 6 to 12 years of age suffering from pulmonary (moderately and far advanced) tuberculosis in various stages of activity. All patients had a definite diagnosis and a thorough work up including tomograms and bronchograms whenever it was necessary in order to ascertain the type of pulmonary lesion. All patients had been observed in the hospital for periods ranging between 1 month and 3 years. The handling of patients was in charge of a nurse who placed the child behind the fluoroscopic screen. The observers were not allowed to see or identify the child until the fluoroscopic examination was over and each observer had recorded his observations independently. The total radiation time was limited to less than 30 seconds in each case.

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* From the Department of Pediatrics of Alejandro Ruiz Soler Sanatorium and the School of Medicine of the University of Puerto Rico.

Presented before the monthly staff meeting of the Alejandro Ruiz Soler Sanatorium, May 1956.

Results:

Graph I shows the types of pulmonary lesions observed in each patient on roentgenograms including PA and lateral films as well as bronchograms and tomograms whenever it was necessary.

Enlargement of the lung hilus or mediastinum, homogeneous densities, deviations of the trachea, mediastinum and diaphragm, and fluid were always detected. Most of the densities that were not homogeneous, described in the graph as feathery infiltrates, were detected. On the other hand, a case of miliary tuberculosis was completely missed. This is not included in the graph. Many of the calcifications, particularly the smaller ones, were not visible in the fluoroscope. Cavities were missed several times. The type of cavity missed was frequently thin walled and in one case it had been classified by the radiologist as a bulla.

Discussion:

Most of the pulmonary lesions in children can be detected with the fluoroscope. Children's thin thoracic walls and narrow chests facilitate visualization of their lungs. Pathologic changes in the moving and flexible structures surrounding the lungs are particularly easy to detect. On the other hand, one should not rely on the fluoroscope when one suspects miliary lesions, thin walled cavities or small calcifications.

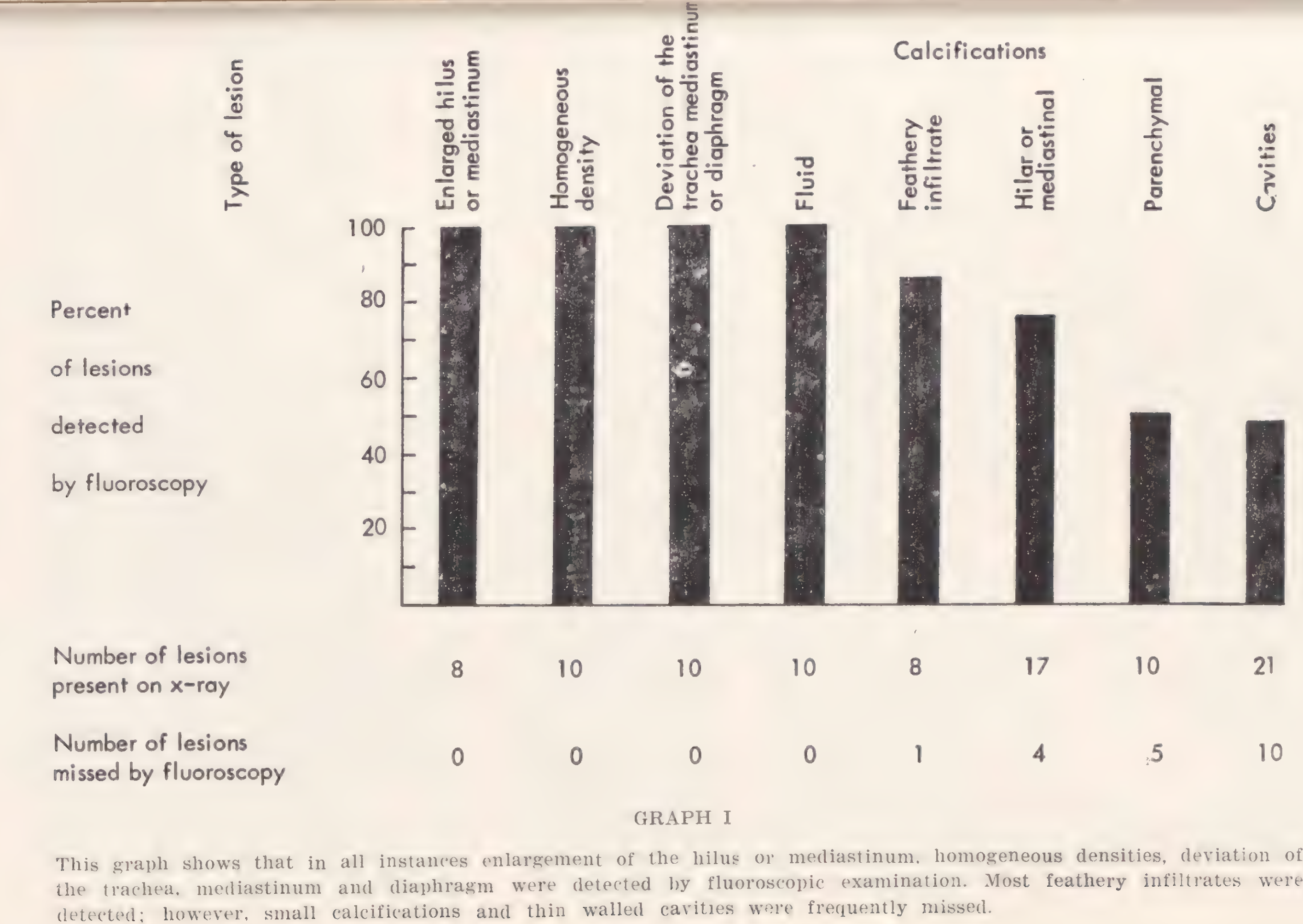
Hetherington and Flahiff[2] in a study of 347 children studied by fluoroscopy and roentgenography were disappointed in the failure to detect by fluoroscopy small and scanty apical involvement limited to the lung above the clavicle. However, they encouraged the use of the fluoroscope for detection of more advanced lesions.

It should be kept in mind that the value of a fluoroscope is greatly increased when the proper technic is used. The paper by Felson[3] on this subject is an excellent one.

SUMMARY AND CONCLUSIONS

This study was designed for the practitioner who has a fluoroscope in his office, but lacks facilities for obtaining X-rays of the chest.

Children suffering from tuberculosis whose pulmonary lesions were thoroughly known were selected for the study. The observers did not know the identity and type of lesion of the patients. It was possible to diagnose the type of pulmonary lesion in most of the cases by means of the fluoroscope. However, in several instances



GRAPH I

This graph shows that in all instances enlargement of the hilus or mediastinum, homogeneous densities, deviation of the trachea, mediastinum and diaphragm were detected by fluoroscopic examination. Most feathery infiltrates were detected; however, small calcifications and thin walled cavities were frequently missed.

thin-walled cavities and small calcifications were missed. In one instance miliary tuberculosis was invisible in the fluoroscope. These limitations should be kept in mind whenever the fluoroscope is used for diagnosis of pulmonary lesions.

RESUMEN

Este informe va dirigido al médico que posee un fluoroscopio, pero carece de facilidades para tomar placas radiográficas.

Se ha intentado hacer una justipreciación de la importancia del fluoroscopio en el diagnóstico de lesiones pulmonares en niños. Se hicieron observaciones fluoroscópicas en niños enfermos con tuberculosis hospitalizados, bajo observación desde un mes hasta tres años. Todos habían sido estudiados repetidas veces con radiografías del tórax además de tomogramas y broncogramas cuando fué necesario para hacer un diagnóstico correcto de la lesión pulmonar.

Fué posible hacer el diagnóstico correcto, usando el fluoroscopio, en la mayoría de los casos. Sin embargo, en otros no fué posible identificar cavernas de paredes finas, pequeñas calcificaciones y tuberculosis miliar. Se hace hincapié en el hecho de que el fluoroscopio tiene estas limitaciones de importancia para el que lo usa como único medio de diagnóstico.

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INDUCTION AND STIMULATION OF LABOR*

*WILLIAM F. FINN, M.D.***

The life of an Obstetrician revolves around labor. Quick, easy labors facilitate delivery and permit safe obstetrics. Labor, however, is not always quick and many times not easy. Labor must frequently be encouraged. Many times it must be started. The various methods of starting labor fall into two general classifications:

1. Medical Methods
2. Surgical Methods

Some of the methods which are now out-moded include castor oil, quinine, solution of Ergot, Pituitrin, various intra-nasal and intramuscular injections, bougies, catheters, and Vorrhees' bags. Quinine is ineffectual, Ergotrate is dangerous from the viewpoint of increased tonus and tetanic contractions of the uterus. Castor oil has been gradually discarded because of it's propensity for producing intestinal cramps without producing uterine contractions. Pituitrin has been gradually out-moded because of its hypertensive effect. The intra-nasal and intra-muscular methods have fallen into discard because of the inhability to control temporary overdosage. Bougies, catheters and bags are alike in their ability to cause infection. These various methods have resolved themselves into two simple techniques. One, the medical approach, consisting of a warm enema and the use of Pitocin intravenously and the surgical method, consisting of rupture of the membranes. There appears to be great dispute how to pronounce Pitocin whether it should be called "Pi-tò-cin" or "Pït-o'cin". For simplicy, let us forget the trade name and call it by its chemical name of oxytocin.

INDUCTION OF LABOR

Indications

The indication which exists for induction of labor may be divided into medical indications and indications of convenience.

There is no question about the need for medical indications. The patient with severe pre-eclampsia who is not responding to

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** Assistant Professor Obstetrics and Gynecology, Cornell Medical College; Associate Attending Obstetrician and Gynecologist New York Hospital; Director, Obstetrics and Gynecology North Shore Hospital, Manhasset, New York.

medical regimen must be delivered either by Cesarean section or by induced labor. Clinical judgement determines which course to follow. The diabetic patient presents the same problem. The mother with the Rh negative blood and increasing anti-body titers presents a similar problem. A further advantage here, is that the laboratory work on the baby such as Rh determination, the Coomb's test, Bilirubin determination, presence of normoblasts, hemoglobin, blood type can be done in the daytime when laboratory technicians are available. Also, it is easier to obtain blood for exchange transfusions at this time of day. There are other medical indications accepted by some. Such as, the induction of premature labor to avert Cesarean section for cephalopelvic disproportion. This places unnecessary hazard on the fetus and is a relic of the days when Cesarean section mortality was very high. Another indication accepted by some, is the presence of premature separation of the placenta. The fetus may be lost during this induction, the uterus cannot be inspected to determine whether it is of Couvelaire type. Accordingly, it seems better to delivery such patients by Cesarean Section.

So much for medical indications for induction of labor. Now we come to the more debatable: elective indications. The induction which is done for the convenience of the obstetrician, so that he knows that he can be present while the patient is laboring. The induction is also done for the convenience of the patient. The patient who has had a history of previous short labors and is worried about transportation to the hospital, particularly if her home is some distant from the hospital. Another patient who is very grateful for the invitation that labor be brought on, is the patient who has had interrupted labor for several days or even several weeks. Such a patient has frequent contractions which may or may not be painful. She attains a partial dilatation of the cervix as determined by sterile vaginal examination performed in the doctor's office. Such a patient, in the final episode of labor may be very precipitate and hence is probably better treated by bringing her into the hospital for an elective induction.

Contraindications

There are both absolute and relative contraindications to induction of labor. The absolute contraindications include:

1. The contracted pelvis with cephalopelvic disproportion.
2. Sensitivity to oxytocin.
3. Marked prematurity
4. Fetal distress

Cephalopelvic disproportion cannot be overcome by driving the head into a resistant bony pelvis which is too small to permit delivery. Such a procedure is fraught with peril to the baby from birth injury due to intracranial hemorrhage. It further, increases the possibility of retraction ring with rupture of the uterus in a later phase of labor. Sensitivity to oxytocin can be manifested by marked tetanic contraction of the uterus with intrauterine death of the baby due to interference with fetal circulation. The baby with marked prematurity, with increased fragility of soft tissue, blood vessel, bone is much more prone to injury and further, when born, is less ready to cope with the manifold problems of extra-uterine existence than his more mature brother. Lastly, most of the intra-uterine conditions which cause fetal distress are aggravated by the use of oxytocin. Fetal distress due to occult prolapse of the umbilical cord can readily be converted into fetal death due to compression of the cord between the head and the bony pelvis. Fetal distress due to partial premature separation of placenta may be readily converted to intrauterine fetal death by extension of the retroplacental hematoma so as to interfere with the passage of blood through the umbilical cord to the fetus.

The relative contraindications to inductions of labor include:

1. High station of the head
2. Previous incisions in the uterus
3. Increased tension of the uterus
4. Abnormal bleeding
5. Parity greater than five
6. Age above 35.

The high station of the head presents a relative contraindiration to the use of oxytocin because of the possible dangers of injury to the fetus in the process of driving the fetus into a normal location in the pelvis. However, at the present time, many competent obstetricians recommend the use of oxytocin in a trial of labor. However, they safeguard the fetus to a certain degree by insisting on pelvic x-rays prior to the attempted induction or in the case of the multipara by a careful appraisal of her previous labors. A high station of the head is frequently associated with a large size baby or with a baby in an abnormal presentation. The abnormal presentation may be partial deflexion of the head and a possible brow or face presentation or a posterior position which is likely to result in long difficult labor. For these reasons, it is thought better to carefully evaluate all featuses whose heads are high prior to an induction of labor. Previous incision into the uterus, whether by Cesarean sections, hysterotomy, or extensive myomectomies with direct entry into the uterine cavity, frequently

so injure the wall of the uterus, that rupture may occur even without labor and is more likely to occur if forceful labor such as sometimes follows oxytocin induction, should occur. Inasmuch as it is impossible to determine in advance which uteri will rupture, it seems most desirable not to induce any patient who has had a previous incision in the uterus. The uterus which is distended far beyond its normal size whether by hydramnios, by the presence of twins, or by a large baby, is a potentially dangerous uterus inasmuch as forcible contractions may increase the probability of rupture. Hence, a uterus which is over distended should be subjected to oxytocin induction in a very careful fashion with an extremely slow start and careful evaluation throughout the process of labor. Occasionally, however, in the case of hydramnios after rupture of the membranes and a slow drainage of fluid, oxytocin may be started with very beneficial effect. A similar situation exists in twins. Though many obstetricians advocate the use of oxytocin induction for cases of premature separation, this seems undesirable because of the possibility of increasing the amount of bleeding during the prolonged induction, of having compression of the fetal cord by an expanding retroplacental hematoma, thus resulting in in-uteru fetal death. Accordingly abnormal bleeding seems to be a relative contraindication to the induction of labor. The woman who has become a grand multipara after having 5 babies, may develop increasing fibrosis of her uterus. This in turn may result in the rupture of the uterus in a very forcible labor such as the induced labor of oxytocin. Some authorities have even thought that three babies are contraindication to the use of oxytocin. However, careful use of the drug in the judicious manner will not cause trouble. A similar line of logic is used in inductions on women over 35. It is thought that a certain resiliency of the uterus has been lost, which makes these women more prone to rupture of the uterus. However, judicious, careful use of oxytocin will not hurt such a woman.

Evaluation

Once induction has been decided upon, it behooves the obstetrician to carefully evaluate his patient for her suitability for delivery. If he should decide that she is not suitable for vaginal delivery by induced labor, Cesarean section should be done. If, however, he decides that delivery should be done by the vaginal route, he should evaluate the patient regarding her cervix, regarding her pelvis, regarding the presentation of the fetus and the station of the head. The cervix is evaluated for its readiness for labor.

The length of the cervix determines its degree of effacement. A length of not more than two cm. is most desirable, whereas further shortening of the cervix to approximately a ½ cm. or one cm. is even more desirable. This effacement of the cervix is an indication that the lower uterine segment is in the process of formation and thus, in an anatomical sense, is ready for labor. The dilatation of the cervix is also helpful in determining its readiness for labor. A cervix which is dilated to one or two cm. is much more ready for delivery than one that is closed. A soft cervix is most desirable as indicative of the various colloidal changes which occur in the cervix prior to natural or induced labor. A point which is not often stressed, but which helps in determining the readiness for labor is the position of the cervix. It is very rare that a cervix which is posterior and sitting on the sacrum is ready for labor. Usually the cervix swings forward and is directly in line with the axis of the vagina before it is ready for labor. The length, dilatation, consistency, and position of the cervix are best determined by vaginal examination during the last weeks of the ante-partum course and just prior to admission. Rectal examinations can be most misleading in trying to evaluate the readiness of patients for delivery. The evaluation of the bony pelvis can be done by physical examination and by pelvic x-rays. Physical examination detects any gross abnormality of the pelvis and permits reasonably accurate measurements of the pelvis. Pelvic x-rays help particularly in the primigravida in whom adequate pelvic examination may not be possible. The lateral film showing the relationship of the baby's head to the pelvis is the most valuable of all the films taken. The presentation of the fetus can be determined by physical examination. The descent of the head is readily determined by rectal or vaginal examination. Engagement, degree of flexion, can be similarly determined. Preferably induction is not done until the baby's head is engaged at the spines or at most is at least one centimeter above the spines.

Technic

After these various criteria have been assessed, the patient is now ready for induction of labor. The scheduling of such a patient is becoming more and more like scheduling a patient for a gynecological operation or a Cesarean Section. The secretary on the delivery floor assigns a time. Patients whose inductions are being done for medical reasons are granted priority, patients who are being induced for reasons of convenience are instructed that it may be necessary to cancel their induction if the delivery

floor is busy at the time originally scheduled. The patient enters the hospital either the morning of the induction or else the night before. She receives no breakfast. She is shaved. Hemoglobin and urine are tested. General physical examination is done. Then at this time, she is once again reevaluated for the last time regarding her suitability for induction. Station of the head, the presentation of the fetus, its size, the type of pelvis, the readiness of the cervix all enter into the evaluation which is summarized on the enclosed record, which is used at North Shore Hospital, Manhasset, N.Y. (Fig. 1.)

Figure I

USE OF OXYTOCIN IN LABOR

Name of Patient ______________________________ Number of Patient __________

Date ________________ Induction ________________ Stimulation ________________

E. D. C. __________________ Parity ____

Reason for Oxytocin ______________________________

Pre-induction Evaluation:

Cervix: Length ______ Dilatation ______ Position ______ Consistency ______

Presenting Part: Vertex ___________________ Breech ___________________

Station: Floating ______________________

Dipping ______________________

Minus 3 ______________________

Minus 2 ______________________

Minus 1 ______________________

Minus 0 ______________________

Plus 1 ______________________

Plus 2 ______________________

Plus 3 ______________________

Membranes: Intact ___________________________ Ruptured ______________________

Pelvic Series: Done _______________________ Not Done _______________________

Estimated Size of Baby ___________________________

Dosage of Oxytocin ______________________________

Time Started _____________________________________

Time Ended ______________________________________

Amount of Solution Used __________________________

Amount of Oxytocin Used __________________________

Type of labor ___________________________________

Complications ____

____

Mother ____

Fetus ____

The use of oxytocin is restricted to the doctor who has full privileges in obstetrics and gynecology — i.e. the doctor who can perform a Cesarean Section, who can evaluate tonus of the uterus, who can perform difficult midforceps deliveries. This doctor must be in attendance at the hospital. His presence on the delivery floor is mandatory. If he leaves the delivery floor, the induction is stopped. The obstetrician should, preferably, be in the delivery room with the patient. He should observe the fetal heart, the blood pressure, the length and intensity of uterine contractions, and should, at various times throughout the induction, feel the fundus to determine its consistency in an effort to pick up approaching tetany.

Adjuvants

Some of the aids to the successful induction of labor are:

1. A warm soap-sud enema which cleanses the bowel and helps stimulate uterine contractions.

2. Rupture of the membranes is most desirable. The timing of the procedure depends upon the type of induction.

In an induction for medical reasons when the patient is not quite suitable for induction but may require several hours to efface the cervix, it is better to refrain from rupturing the membranes until it is certain that the induction will go on successfully. In other words, when the cervix is several centimeters dilated and the membranes are bulging, it is most desirable to rupture the membranes inasmuch as the drainage of fluid permits the uterine muscle to contract on a shorter length and so work more efficiently. Prior to rupture, the membranes are carefully felt in an effort to detect vasa previa. The position, and station of the head are both determined. The presentation is again checked to be certain that it is a vertex and not a breech. An effort is made to pick up the various elements that make for compound presentation, arm, foot or an occult prolapse of the cord. The membranes are ruptured by using a thin plastic sound, similar to a knitting needle. The fluid is emptied slowly, the fingers are kept in the vagina to detect any prolapse of cord and the presenting part is pushed down into the pelvis as deeply as possible. The color and consistency of the amniotic fluid is checked to detect the presence of meconium which might be indicative of intra-uterine distress. The medical induction is now started by the insertion of a #18 needle into a vein in such a fashion that the vein is cannulated. A vein far away from one of the movable joints in the arm is selected so that if later on the patient should move, she will not

disengage the needle. A bottle of 5% glucose and water is attached. A bottle of 5% glucose and water with 5 international units of Oxytocin is then prepared. This results in a dilution of 1 international unit of Oxytocin to 100 cc's of glucose and water. This second bottle of solution is then inserted in the rubber tubing of the first control solution of glucose and water. The first bottle which is known as the control can then be used at any time to keep the vein open if the oxytocin solution must be turned off. A three-way stopcock, was formerly used, but proved to be too cumbersome. It is far preferable to speak of international units which are standard rather than in terms of minims which may vary. The oxytocin solution is then started in a slow fashion of approximately 3-5 drops per minute. This is virtually an intra-venous sensitivity test, since the patient is observed for marked increase of the tonus, prolonged contractions, abdominal pain, development of rash, appearance of dizziness, the development of hypertension, and variation of the fetal heart. During this crucial 15 minutes of the start of the induction, the obstetrician is in constant attendance on the patient observing the various vital signs. Contractions, length and frequency are noted, the fetal heart is checked, the patient's pulse and blood pressure are taken. If no problems develop during the time, the oxytocin solution is gradually increased to about 10 to 12 drops per minute and continued in this fashion until the patient has started in normal labor.

The effort in induction is to mimic normal labor as closely as possible. The goal is to have contractions which are similar to normal non-induced contractions in that they start slowly, build up to an apex and then gradually recede. If at any time there are very strong contractions, the solution is gradually decreased until the contractions are within the range of normal intensity and frequency. These precautions should destroy the possibility of tetany. Occasionally, tetany will suddenly appear; the uterus will become rock-hard. At that time oxytocin is immediately turned off, while the control solution is started. Two cc's of 50% Magnesium sulfate is injected into the vein to decrease the intensity of the contractions. Open drop ether is administered to the patient until the uterus relaxes. Constriction or retraction rings are checked for at frequent intervals. These are usually readily felt except in the obese patient, at which time they cannot be detected well by abdominal palpation. Then the lack of progress, the soft flabby cervix which is not completely retracted, permit the thought of ring to be entertained. Fetal distress is an indication to stop oxytocin. It may indicate prolapse of the cord which can be readily detected by vaginal examination. It may indicate pre-

mature separation of the placenta, which is usually accompanied by external bleeding. It may merely indicate a relative interference with the blood supply of the fetus due to the force of labor. This can be corrected by decreasing the dosage of oxytocin. Signs of shock such as tachycardia or hypotension, mean that oxytocin must be discontinued. An immediate vaginal and abdominal examination must be done to rule out the possibility of rupture of the uterus or massive premature separation of the placenta. Hypertension rarely occurs with oxytocin administration, but when it does occur, it is best treated by intravenous sedation, such as 100 mg. of seconal. Presence of rash may indicate a sensitivity to oxytocin and hence alerts the obstetrician to be on the lookout for other signs of sensitivity. Oxytocin may be continued in a careful fashion, if rash develops.

Analgesia and Anesthesia

The patient who is receiving intravenous oxytocin is to a certain degree in a phase of controllable labor which the obstetrician knows will continue even though analgesia and anesthesia are given. Hence it is well to administer some analgesia prior to the start of the induction. This may consist of demerol or morphine in an appropriate dosage. These are usually accompanied by atropine or scopolamine. The former is used for the drying effect on the respiratory passages, the latter for this effect plus the amnesia which may occur. Inasmuch as the patient already has a needle in her vein it is very easy to give these various medications into the rubber tubing so that they are administered intravenously. Medication given in this fashion takes effect in approximately two to three minutes and reaches its peak in about 20 to 30 minutes and then has pretty well worn off in approximately an hour. In contrast intramuscular medication begins to take effect in about 15 minutes, reaches its peak in about an hour and then wears off in approximately 2 hours. The intravenous route is particularly desirable for use in a quick fashion near the end of labor. Usually morphine (nalline) is administered in 5 mg. dosage into the rubber tubing as the patient is taken to the delivery room. This acts as an antagonist to the respiratory depression of morphine and so aids the fetus in establishing respiration sooner. Various forms of regional anesthesia may be used in conjunction with the oxytocin induction. Caudal or epidural anesthesia started with dilute concentrations of Zylocaine in the range of 0.25% may provide very effective relief of contractions for several hours. The dosage of Zylocaine can gradually be increased to 0.5, 0.75,

1% and then on up to a concentrated solution of 2% immediately prior to removal to the delivery room. 1% Procaine can be used as pudendal block while general anesthesia, cyclopropane or ethylene may be used if the patient should desire to be placed asleep.

Conduct of Labor

Conduct during labor is essentially the same as when the patient is not induced. The various vital signs, the fetal heart, and the contractions are checked at frequent intervals. Internal examinations are performed to determine the station of the head and the dilatation of the cervix. Sedation, fluid, anti-microbial therapy are given as indicated. When a patient is ready for delivery, oxytocin is usually slowed, the patient is transferred to the delivery room and delivery accomplished either by spontaneous or operative methods. Once the fetus is delivered, the oxytocin is restarted. Once the placenta has been expelled the patient is given ergotrate intramuscularly to sustain action in keeping the uterus tight. Any delivery after oxytocin requires inspection of the cervix because the last stage of the cervical dilatation may be referred to Duhrrsen's incisions performed by oxytocin rather than by a scissors. The cervix is grasped by sponge sticks and inspected. Tears, when found, are usually posteriorly at 6 o'clock. They usually are not bleeding and can be repaired with interrupted or continuous chromic catgut suture. After the episiotomy has been repaired by routine fashion the patient is placed in a postpartum recovery room. For one hour after delivery vital signs are observed, fundus is observed for contractions and oxytocin is continued in a slow fashion. If oxytocin is discontinued at the time of delivery, atony of the uterus frequently results causing increased bleeding in the so called fourth stage of labor.

Complications

Such a method of induction eliminates some of the dangers and complications which may occur. These may be regarded on the basis of the mother and the fetus. From the basis of the mother, there are tetany and the formation of constriction or retraction rings which may progress to rupture of the uterus, either partial or complete. The mother may experience hemorrhage due to premature separation of the placenta or she may have amniotic fluid infusion or may show extensive tears of the cervix. The fetus may be smothered at birth by marked compression of the placental sponge with interference with the proper circulation,

or may be born listless and show definite evidence of intracranial hemorrhage. This potent drug must be used carefully to avert such complications.

STIMULATION OF LABOR

Labor which has started of its own accord frequently becomes sluggish or inert. The contractions may have been infrequent and of poor intensity from the very beginning of the labor, the so-called primary inertia, or contractions which once were forceful and frequent, have now become very poor in quality, secondary inertia. In general, contractions which occur at intervals of more than 5 minutes and which last less than 30 seconds are indicative of sluggish inert labor. This is further manifested by lack of progress. There is a failure in the descent of the head, the station remaining at its original position. This may be associated with posterior position of the baby's head. The cervix instead of dilating becomes progressively thicker and more edematous. Some of these delays may be due to over sedation or to regional anesthesia. Occasionally, the delay may even occur in the second stage of labor. Full dilatation has occurred and yet the patient has infrequent, powerless contractions. Under such circumstances, the patient is evaluated to rule out cephalopelvic disproportion, abnormal presentation or unsuspected large size of the fetus. The patients must be carefully evaluated for these contraindications to the use of oxytocin. Oxytocin should not be started if the labor is of normal quality. It should not be started if the uterus appears to have tonus than the average uterus. It should not be started in the presence of cephalopelvic disproportion. Whether it should be started for the abnormal presentation is debatable. Certainly it should not be started for a face or a markedly deflexed head though it may be used with care in a breech presentation. It should not be started in the presence of fetal distress, because we may compound the already existing injury to the fetus. Patients who require stimulation of labor usually must be evaluated further. The patient may be dehydrated. This can be readily corrected by intravenous solutions of glucose and water. The patient may show acetone in the urine; this is corrected by the presence of glucose. Infection is combated by therapy. A patient who is in a marked panic state is best handled by heavy sedation which completely removes all sensation. The exhausted patient is best treated not by stimulation, but rather by sedation and then, if spontaneous labor does not result, by careful stimulation. The uterus which is more or less paralyzed and atonic is frequently

helped by rupture of the membranes. The patient who is over-sedated is best treated by effective uterine stimulation with oxytocin. The conduct of labor, analgesia and anesthesia, conduct of delivery and postpartum care are the same as when labor is induced.

SUMMARY

Oxytocin is still in its experimental stages. There are many areas of applicability and many disputed points in its use. Some of these include:

1. Should the induction be an interrupted induction or an all-out induction? This applies particularly to the patient who requires medical induction but is unsuitable because of the length and consistency of the cervix.
2. Should a "trial-labor" be given with the use of oxytocin or should Cesarean section be done instead?
3. Should oxytocin be used when the vertex is floating in a multipara with a good pelvis?
4. Can oxytocin be used to correct the occiput posterior position and by means of producing better labor help the spontaneous rotation to the anterior position?
5. Is it safe to use oxytocin in the case of face, brow, twins or breech?

None of us know the answers to these questions at the moment. We have in our hands a powerful drug which used in a judicious manner can immeasurably aid the course of labor. This means careful selection of patients, frequent checks of vital signs, and detection of possible complications in their very early stages. By so using oxytocin, we will imitate spontaneous labor as much as possible. Since oxytocin is being used and will be used with increasing frequency, we must learn to use it properly.

USE OF PIROMEN IN OPHTHALMOLOGY*

FINAL REPORT WITH ADDITIONAL CLINICAL STUDIES

ROBERTO BUXEDA, M.D.
San Juan, P. R.

This is a report on clinical observations and studies based on the results obtained in the treatment of eye diseases with Piromen,** a new drug. As far as I have been able to ascertain this is the first report on the use of this drug in the therapy of eye conditions.

During the past two years I have treated some 60 patients with different ocular diseases with Piromen. The present report is based on the results obtained in treating 27 cases which were followed up by me closely. The results with this drug in treating a number of patients with a wide variety of diseases of the eye have been, in my opinion, satisfactory enough to warrant the writing of the present thesis. I acknowledge the fact that the number of cases of each eye disease treated has been small, indeed. In some diseases the number has consisted of only one case; in other diseases it has consisted of as many as five cases. The uniformity of results in the latter permits a more or less accurate evaluation of the drug for that particular condition. However, the over all results have been consistent and satisfactory enough to arrive at the conclusion that Piromen has definite value in ophthalmology and that trial of the drug by other ophthalmologists is indicated.

Kronfeld[1] was one of the first ophthalmologists to talk about the use of Piromen in the treatment of eye diseases. According to him the drug is a satisfactory pyrogenic agent. The first trials with the drug were aimed at the production of hyperpyrexia for the therapy of ocular conditions in much the same way that typhoid vaccine has been used for several decades. Extensive clinical use of Piromen by different authors[2,3,4,5,6] in the treatment of food allergy, perennial respiratory allergies, skin allergies and other dermatological conditions in subpyrexia doses have established the fact that hyperpyrexia is not essential to bring about good therapeutic results. Since the author[7] has shown that in the treatment of eye diseases with typhoid vaccine infusions the production of a high fever is not necessary for beneficial therapeutic effects

* From the Section of Ophthalmology, School of Medicine, University of Puerto Rico, San Juan, P. R.

** Supplied by Baxter Laboratories, Morton Grove, Illinois, through the courtesy of Dr. N. M. Nesset.

I decided to use Piromen in doses that would not lead to the production of fever. In most of the patients treated 10 gamma doses of this material did not produce any fever. In a few cases a very slight elevation of temperature occurred.

DEVELOPMENT OF PIROMEN

Some observers have repeatedly noted beneficial therapeutic results incidental to the intravenous administration of fluids. Investigation into this matter led to the discovery of the fact that these beneficial effects derive from the presence of a contaminant in the fluids used for venoclysis. This contaminant is pyrogenic in nature and has been shown to be the cause of the chills and fever which are frequently observed during the intravenous administration of fluids. Attempts to isolate the material responsible for these reactions led to the preparation of Piromen by Nesset and his colleagues.[8]

NATURE OF PIROMEN

Piromen is a pyrogenic substance obtained from Pseudomonas aeruginosa. This material has been shown to be a relatively nontoxic and a non-aphylactogenic bacterial substance.[9,10] Pharmacologically it is a highly reliable pyrogen which has very little toxicity. Nesset and his associates[8] have shown that it is non-protein in nature. Several groups of investigators[9,10,11,12,13,14,15] have shown that pyrogens are polysaccharides. These agents have been termed polysaccharides primarily because reducing sugars are obtained on hydrolysis. The nature of all the component reducing sugars is not as yet well known but hexosamine has been fairly constantly detected. The only other sugars found in the Pseudomonas concentrate by Nesset and his co-workers[8] in their experimental work with this substance were desoxyribose in desoxyribonucleic acid and ribose in ribonucleic acid. According to these investigators the nucleic acid do not appear to be esential for the production of fever. Hexosamine appears to be the only reducing sugar essential for fever production according to these authors.

Since it has long been known that artificial fever, produced physically as is the case with the hypertherm cabinet or chemically as is the case with the intravenous administration of killed typhoid organisms or malarial parasites has therapeutic value, research groups immediately embarked upon the idea of using Piromen for the artificial production of fever. With this view in mind it was used extensively by Lonsen and Liebert[16] in the treatment of neurosyphilitic patients and by Kierland and Kulwin[17] in the treatment

of skin diseases. It was also used by other investigators[18 19 20 21 22] in the treatment of hypertension in doses large enough to produce hyperpyrexia.

It soon became evident to some of the research men working with Piromen that relatively small doses which were not followed by any marked constitutional symptoms gave better results. Thus, Randolph and Rollins[23] used the material in subfebrile doses for the treatment of perennial allergic symptoms. The same procedure was followed by Wittich[3] in treating perennial respiratory allergies and by Fitch and Washburne[24] in treating mentally depressed patients. Guerrieri[25] and Zindler[26] likewise, used the small dosage schedule in treating neurodermatitis and food allergy respectively.

In treating my series of eye cases I have decided to follow the latter method of administering subfebrile doses since it has been my experience[7] in administering typhoid vaccine in a continuous intravenous infusion that the production of a high fever is not absolutely necessary to bring about beneficial therapeutic results.

ACTION OF PIROMEN

The intravenous administration of Piromen is characterized by a marked mobilization of the reticulo-endothelial system, by a transient leukopenia and eosinopenia followed by a neutrophilic leukocytosis, by a lymphoid tissue hyperplasia and by a stimulatory action on the adrenal cortex. Elevation of the body temperature occurs only when given in doses larger than will be recommended in this article. As in foreign protein therapy in general, the beneficial therapeutic effects of Piromen are probably not due to any particular one of these reactions but rather to a combination of these. According to Chambers and his co-workers[15] the fever response to Piromen when it occurs is mediated through the central nervous system.

According to Lonsen and Liebert[16] microscopic examination of the organs of animals killed after these had been subjected to long and massive experimental administrations of Piromen did not show any signs of permanent damage. These authors concluded that this material effects a mobilization of the general body defenses within a wide range of safety. It appeared to them that Piromen in large doses seemed to be safer than malaria or typhoid vaccine therapy in persons over 60 years of age.

Windle and his colleagues[14] have also likewise studied extensively the reaction of tissues in experimental animals to both ordinary and massive doses of Piromen with similar results. This last group of investigators found, however, that significant his-

tological changes occurred in and were limited to the kidneys, suprarenal glands, lymph nodes, bone marrow and spleen. The livers of some of the animals showed small areas of leukocytic infiltration, minute foci of necrosis or minute granuloma formations. This research group did not find any significant difference in the livers of the animals receiving Piromen from those receiving an extract of Eberthella typhosa and from those of the control animals.

These authors, therefore, assumed that the liver changes were unrelated to the administration of the different materials.

Examination of the suprarenal glands of the rabbits that had received recent administrations of Piromen in the experiments of Windle and his coworkers[14] showed changes which were most marked in the zona reticularis of the cortex. The cells here were swollen, full of droplets and even vacuolated and the zone was thicker than in the control animals. The glands of the rabbits receiving the most prolonged massive dosages were of relatively normal appearance but contained clumps of swollen cells in the reticularis. The picture was suggestive of an acceleration of cortical secretory activity. These observers noted that the most consistent alterations occurred in the spleens, lymph nodes, and bone marrow of the rabbits. They summed up their findings in these organs by stating that the lymphoid and myeloid organs showed varying degrees of hyperactivity, hyperplasia, and, sometimes metaplasia.

Favorite and Morgan[27] have also reported histological changes after the administration of other pyrogenic substances. They have shown that alterations in the proportions of the white blood corpuscles commonly occur. These investigators, for example, found a granulocytic leukocytosis following the administration of a pyrogenic extract of Eberthella typhosa.

METHOD OF ADMINISTRATION AND DOSAGE

The administration of Piromen does not impose the severe restrictions on the patient that the intravenous administration of typhoid vaccine does. The patient can continue leading his usual normal every day life while the treatments are being given. The symptoms produced by the drug are not incapacitating in any way in the ordinary case. His meals are not interfered with and he does not have to go to bed.

The treatments can be administered on an ambulatory basis.

The preparation should be given intravenously. It can, however, be given subcutaneously.

The intravenous administration of Piromen is usually followed

in 45 minutes to an hour by a chill. A slight headache is also a part of the ordinary reaction. The patients may also complain of myalgia and rarely of nausea. Most of these symptoms are easily relieved by aspirin, the use of which is encouraged at four-hour intervals for the comfort of the patient. This drug does not appear to interfere with the beneficial effects of Piromen. The initial dose of Piromen for the treatment of eye diseases should be five gamma. The following day or two days later the dose is increased to 7½ gamma. Subsequent administrations should consist of 10 gamma of the material every other day until the treatment is completed. The number of treatments depends on the individual case and on accurate observations at regular intervals. Whereas certain cases may require as little as three or four injections, others may require as many as 25 or 30.

RESULTS

I Acute Iritis (2 cases)

Case 1: This case showed a moderate ciliary flush and a +3 aqueous flare with numerous cells in the anterior chamber. Two days after the intravenous administration of 5 gamma of Piromen a slight ciliary flush and hardly any aqueous flare were noted. The patient volunteered subjective improvement and no further treatment was considered necessary.

Case 2: This case showed marked ciliary flush, tenderness on palpation of the eye and a +1 aqueous flare. After three injections of Piromen administered every three days the patient was asymptomatic and only an occasional cell was noted in the anterior chamber.

II Traumatic Iritis (1 case)

This patient had been hit in the eye with a piece of rope. The following day he noticed redness, pain, photophobia, lacrimation and dimness of vision. Examination of the eye revealed intense ciliary flush and a semidilated pupil which reacted sluggishly to light. Slit lamp examination showed numerous pigmented cells moving with the circulation of the anterior chamber and a tear of the sphincter with posterior synechiae at 7 and 8 o'clock.

After the intravenous administration of five gamma of Piromen the number of pigmented cells in the anterior chamber had easily decreased to one half; the ciliary flush had definitely decreased and the patient volunteered marked subjective improvement.

III Post operative Iridocyclitis (3 cases)

Case 1: This patient developed post operative iridocyclitis after a combined intracapsular cataract extraction. He complained of seeing a thick cloud moving around in the operated eye.

Examination of the eye showed thick anterior vitreous opacities, which did not permit visualization of the fundus. After nine injections of Piromen the vitreous opacities had all but vanished and the optic nerve head and the rest of the fundus could be visualized without any difficulty.

Case 2: This patient developed an iridocyclitis following a capsulectomy operation. Thick vitreous opacities did not permit visualization of the fundus properly. The visual acuity just before Piromen was started was finger counting at 5 feet which was not correctible. After seven injections of piromen the opacities cleared to a point that permitted visualization of the nerve head and the fundus generally and corrected visual acuity was 20 40.

Case 3: This patient had sustained an anterior dislocation of the lens following trauma several years previously. About six weeks before admission to the hospital he developed severe pain in the eye and was diagnosed as having glaucoma secondary to an anterior dislocation of the lens. The eye was markedly congested and tender.

After extraction of the lens the glaucoma disappeared and instead the eye became extremely soft and severely congested. Ten days postoperatively and noticing that the inflammatory process persisted I started administering Piromen intravenously every other day. After the third injection it was unquestionable that the inflammation had receded considerably and the patient felt greatly improved subjectively. After seven more injections the eye had quieted down and was well on the way to complete recovery.

IV Acute Chorioretinitis (5 cases)

Case 1: This case presented an acute lesion of chorioretinitis near the site of an old healed lesion of the same nature. The visual acuity before Piromen was started was 20 200 just before Piromen was started. After 13 intravenous injections of the drug given every other day the visual acuity (without correction) had improved to 20 50. After this drug was administered 10 times the vitreous opacities had almost completely cleared and the corrected visual acuity (with a + 0.50 cylinder axis 90) was 20 25-2.

Case 2: This patient had a visual acuity of 20 200 just before Piromen was started. After 13 intravenous injections of the drug given every other day the visual acuity (without correction) had improved to 20 50. At this stage the lesion appeared quiescent

and the patient's only complaint was that of seeing a black spot moving around.

Case 3: This was a case of a tiny lesion of chorioretinitis in which the patient complained of a snake-like structure moving around. The visual acuity before Piromen was started was 20 25. After eight injections of the drug the patient's chief complaint disappeared completely and the one single long vitreous opacity noticed at the first examination was no longer visible.

Case 4: This was a case of central chorioretinitis in which there were no vitreous opacities. The visual acuity at the first examination was finger counting at 1 foot. This was not correctible. After nine injections of Piromen the visual acuity improved to 20 200 with correction and after ten additional treatments it was 20 40 with correction. At this stage the macular lesion appeared quiescent there being pigmentation and the foveal reflex present.

Case 5: In this case the lesion of chorioretinitis could not be visualized before treatment due to thick vitreous opacities. The visual acuity was finger counting at 3 feet. After 10 injections of Piromen administered in 3 weeks the vitreous opacities had almost completely absorbed and the visual acuity was 20 200. The lesion was then found to be situated in the macular region.

V Acute Uveitis (1 case)

This patient complained of a thick shadow in his right eye. At examination the only finding was that of moderately thick vitreous opacities. No lesion of chorioretinitis was noted. All laboratory examinations and search for focci of infection were negative. The visual acuity before treatment was started was 20 200. After four injections with Piromen the vision had improved to 20 70 with the pupil dilated with atropine and the vitreous opacities were of the nature of a thin veil. Six additional injections were given and with the last of these atropine was discontinued. Two weeks later the patient's uncorrected vision was 20 25-3.

VI Chronic Uveitis (1 case)

This was the case of a patient with a blind eye who complained of pain in the eye and in whom examination revealed moderate ciliary flush and numerous cells in the anterior chamber. After nine injections of Piromen were given every other day only slight residuals of inflammation remained and the pain was gone.

VII Central Retinitis due to Light (1 case)

This patient had been looking at a strong artificial light when he suddenly noticed a large scotoma in straight ahead vision in one eye. The patient was seen by me 13 days after the onset of the condition at which time examination revealed a large central scotoma extending between 10 to 15 degrees around fixation. Funduscopic examination showed macular edema and no foveal reflex. The vision was 20/400 and it was not correctible. After nine intravenous injections of Piromen were administered on alternate days the scotoma disappeared and corrected vision was 20/15-2.

VIII Vitreous Opacities (1 case)

This was the case of a 72-year old woman who complained of seeing cloudy out of her right eye. At examination no evidence of inflammation was found in the anterior chamber but the vitreous showed thick opacities which did not permit good visualization of the fundus. Before starting treatment the visual acuity was 20/200 which was not correctible. After 13 injections of Piromen administered in 26 days the fundus was visualized through a faint vitreous haze and at this time accurate study did not reveal any lesion of chorioretinitis. Vision with correction at this time was 20/30 + 3.

IX Retrobulbar Neuritis (3 cases)

Case 1: This patient had a visual acuity of finger counting at four feet. After 20 injections of Piromen administered in a period of 52 days the vision improved to 20/20-2 (uncorrected).

Case 2: The visual acuity in this patient before treatment was 20/100. After three injections of Piromen administered in a period of six days the vision had improved to 20/40.

Case 3: This patient had a visual acuity of finger counting at four feet with her correction. After five Piromen injections the vision had improved to 20/200 with her correction.

X Alcohol Amblyopia (1 case)

This patient had been on a drinking spell continuously for about one month when he noticed visual loss. There were bilateral central scotomata and the vision in each eye was 20/300. After 17 injections of Piromen the vision in each eye had improved to 20/20 with a small astigmatic correction. The time elapsed from the day of admission to the hospital until the patient was discharged with normal vision was 38 days. No other treatment was given to this patient.

XI Optic Neuritis (1 case)

This patient had a visual acuity of 20 70 before initiation of treatment and 20 50 + 2 after nine injections of Piromen administered in a period of 17 days. The patient discontinued coming to the clinic after this period of time.

XII Endophthalmitis (1 case)

This case developed a white purulent material just behind the pupil eight days after an intracapsular cataract extraction. The eye was markedly congested and the visual acuity was only of light perception. After 22 injections of Piromen given every other day the exudate had absorbed to a point where visualization of the nerve head and the posterior pole of the eye was possible through the membranous strands that remained after the pus was absorbed. After discission of the membrane the fundus was visualized to perfection and it was then noticed that the patient had a scar of old healed chorioretinitis in the macular region. In spite of this vision was correctible to 20/70.

XIII Vitreous Hemorrhage and Hyphema (2 cases)

Case 1: This patient complained of pain on admission. The fundus could not be visualized and the visual acuity was light perception. After 25 Piromen injections the vitreous was very clear and the fundus could be visualized and the vision had improved to finger counting at three feet. This patient volunteered much relief of pain immediately after the first injection.

Case 2: This case was a severe case of vitreous hemorrhage and hyphema in which repeated paracentesis did not help. The patient developed blood staining of the cornea in the affected eye and although the hemorrhage did not appear to be affected by Piromen this seemed to bring about much relief of pain.

XIV Disciform Keratitis (1 case)

This patient's lesion involved the pupillary area of the cornea and the visual acuity before treatment was 20 200. After three Piromen injections the vision improved to 20 100. Further treatment with this drug and with other drugs including cortisone locally did not bring about any further improvement in visual acuity. Two and a half months after the initiation of treatment the vision was still 20 100 and the appearance of the lesion the same.

XV Keratitis (3 cases)

Case 1: This case had a keratitis involving the pupillary area of the left eye. The visual acuity before the institution of treatment was 20/80 and after 14 Piromen injections 20/70 and there remained then only a minimal edema of the cornea, the patient being asymptomatic. I presume that this case had a refractive error; however, I regret that no refraction was done.

Case 2: This patient had an irregularly circular area of keratitis just within the edge of the pupillary area of the cornea which interfered with vision slightly. The involved area stained with mercurochrome and slit lamp examination revealed involvement of the superficial layers of the substantia propia. After three Piromen injections the affected area had healed completely and only an area of opacity remained. At this stage the patient was free of his original subjective symptoms of photophobia and pain.

Case 3: This patient had a marginal keratitis for which he had been treated by another ophthalmologist with cortisone drops and antibiotics locally with no apparent success.

After two Piromen injections the keratitis had healed completely and the patient was virtually asymptomatic.

It should be pointed at this stage that the only other accessory medication in all cases hereby described was atropine locally where this drug has been classically used to prevent the formation of posterior synechiae and to put the eye at rest.

The tabulated results in this series of cases is presented in Table 1.

DISCUSSION

It is conceded that the number of cases included in this report is small. The evidence is however, convincing that Piromen is a valuable new addition to the ophthalmological armamentarium.

Although Piromen is a pyrogenic substance, use of its pyrogenicity has not been made of in the present study. In the doses used in this series of cases the most obvious alterations observed have been on the cellular elements of the blood. Characteristically these alterations include a transient leukopenia followed by a neutrophilic leukocytosis and eosinopenia.

The peak of the total and polymorphonuclear leukocytosis following the administration of Piromen is variable in its occurrence, in some cases occurring two hours after administration (Table 2) and in others occurring as late as eight hours after ad-

ministration (Table 3). The total leucocytosis attains a height of two and a half to three times the initial count. This leukocytosis in all probability plays a role in scavenging away inflammatory products at the site of the ocular pathology. The blood picture is usually restored to normal in 16 to 24 hours after Piromen administration.

Tables 2 and 3 show typical responses to Piromen administration in two of the patients treated in this series. This response is similar to that following stress and the systemic administration of cortisone and adrenocorticotropic hormone (ACTH). It is therefore likely that in addition to the leucocytosis the production of cortisone is an important action of Piromen.

It is also suspected that Piromen brings about or produces an increase in the number of circulating antibodies in much the same way that typhoid vaccine does.

Adrenocorticotropic hormone (ACTH) and cortisone systematically are both drugs not to be tampered with freely due to their inherent dangers. By comparison the accumulating evidence would indicate that we have in Piromen a very promising new therapeutic agent, which possesses many of the beneficial properties of adrenocorticotropic hormone (ACTH) and cortisone but is safe, relatively free from side reactions and readily available.

Piromen has the advantage over cortisone locally that it "works from within". Its effects, therefore, reach all the cells uniformly thereby affecting each cell including the diseased ones through its multiple actions. The penetrability of cortisone locally used is well known, diminishes the farther away the cells are from the anterior surface of the cornea and it is conceivable that many cases of iridocyclitis may not be satisfactorily affected.

Piromen has the advantage over typhoid vaccine that it does not impose any particular restrictions on the patient. Individuals receiving this form of therapy do not require hospitalization, and generally are not unduly uncomfortable. The general malaise, severe headache, nausea and vomiting which accompany typhoid vaccine administration are virtually unknown after Piromen administration.

SUMMARY AND CONCLUSIONS

Piromen appears to be a valuable drug in the treatment of inflammatory conditions of the eye.

This material appears to produce its beneficial effects through the production of a marked leucocytosis which is characterized by a marked increase in the circulating polymorphonuclear cells, through an increase in the circulating antibodies, and through a stimulation of the pituitary-adrenal axis.

Piromen possesses many of the beneficial properties of adrenocorticotropic hormone (ACTH) and cortisone but is safe, relatively free from side reactions and readily available.

This drug has the advantage over typhoid vaccine that hospitalization is not required and that the reaction is much milder.

The therapeutic effects of Piromen occur in the absence of a pyrogenic response. The avoidance of a pyrogenic reaction is a desirable feature in that a series of disturbing side effects are eliminated.

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TABLE 1

RESULTS OBTAINED IN TREATMENT OF EYE DISEASES WITH PIROMEN

Diagnosis and case	*No. of treatments*	*Vision before treatment*	*Vision after*	*Signs and symptoms before tr't*	*Signs and symptoms after tr't*
1. Acute Iritis					
Case 1	1	20/25	20/20	+3 flare	occas. cell
Case 2	3	20/30	20/25	+1 flare	asymptom, occas. cell
2. Traumatic Iritis	1			numerous cells	½ number cells
3. Post operative Iridocyclitis					
Case 1	9			thick opacities	clear, fundus seen
Case 2	7	C.F. 5 ft.	20/40 (corrected)	thick opacities	clear
Case 3	10	blind	blind	severe inflammation	much improved
4. Acute Chorioretinitis					
Case 1	10	20/200	20/25−2 (0.50 x 90)	thick opacities	almost clear
Case 2	13	20/200	20/50 (uncorrected)	thick opacities	only "black spot"
Case 3	8	20/25	20/20	"snake"	"snake vanished"
Case 4	19	C.F. 1 foot	20/40 (corrected)	macular edema, pigmentation	lesion quiescent
Case 5	10	C.F. 3 feet	20/200	thick opacities, fundus not seen	lesion seen in macula

TABLE 1. (Continued)

Diagnosis and case	No. of treatments	Vision before treatment	Vision after	Signs and symptoms before tr't	Signs and symptoms after tr't
5. Acute Uveitis	10	20/200	20/25—3	mod. thick opacities	faint haze
6. Chronic Uveitis	9	blind	blind	pain, many cells	pain gone, little inflammation
7. Central Retinitis due to Light	9	20/400	20/15—3 (corrected)	central scotoma, macular edema	scar
8. Vitreous Opacities	13	20/200	20/30+3 (slight correction)	thick opacities	fundus visualized
9. Retrobulbar Neuritis					
Case 1	20	C.F. 4 feet	20/20—2 (uncorrected)	central scotoma	none
Case 2	3	20/100	20/40	central scotoma	faint scotoma
Case 3	5	C.F. 4 feet (with correction)	20/200 (with correction)	central scotoma	still present
10. Alcohol Amblyopia	17	O.U. 20/300	20/20 (small astigmatic correction)	bilateral central scotomata	none
11. Optic Neuritis	9	20/70	20/50+3		
12. Endophthalmitis	22	Light perception	20/70 (corrected after discission)	exudate behind pupil, fundus not seen	absorbed, fundus seen, healed parafoveal choroiditis

TABLE 1. (Continued)

Diagnosis and case	*No. of treatments*	*Vision before treatment*	*Vision after*	*Signs and symptoms before tr't*	*Signs and symptoms after tr't*
13. Vitreous Hemorrhage and Hyphema					
Case 1	25	Light perception	C.F. 3 feet	pain, fundus not seen	pain relieved after first injection, fundus seen clearly
Case 2	16	blind	blind	pain, vit. hemorrhage, blood staining of cornea	pain relieved
14. Disciform Keratitis	9	20/200	20/70		vision not improved further
15. Keratitis					
Case 1	14	20/70	20/50	photophobia, pain, edema	minimal edema, asymptom.
Case 2	3			corneal edema	ulcer healed, opacity
Case 3	2			marginal corneal edema	healed completely, asymptom.

TABLE 2. — LEUCOCYTIC RESPONSE TO 5 GAMMA OF PIROMEN IN THE CASE OF C. V. R. (KERATITIS)

HOUR	WBC	POLYS	LYMPHOS	MONOS	EOS
*8:00 AM	5,100	52	43	0	5
10:00 AM	13,800	86	12	0	2
12:00	8,300	77	23	0	0
2:00 PM	6,750	70	26	0	4
4:00 PM	6,950	54	29	0	6

* Piromen was administered immediately after the blood sample for the 8:00 A. M. count was taken.

TABLE 3. — LEUCOCYTIC RESPONSE TO 5 GAMMA OF PIROMEN IN THE CASE OF F. C. (POST OPERATIVE IRIDOCYCLITIS)

HOUR	WBC	POLYS	LYMPHOS	MONOS	EOS
*8:00 AM	9,000	60	33	4	3
10:00 AM	9,600	83	17	0	0
2:00 PM	14,500	95	5	0	0
4:00 PM	21,750	95	5	0	0

* Piromen was administered immediately after the blood sample for the 8:00 A. M. count was taken.



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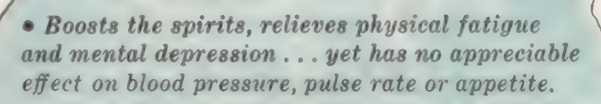
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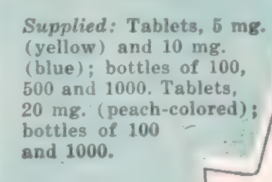
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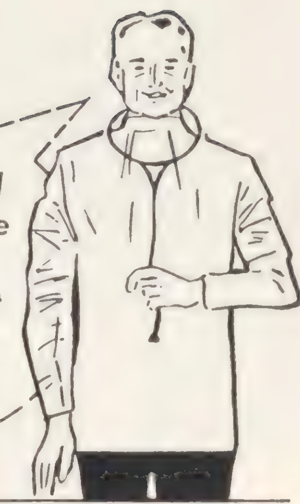
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DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 48 OCTUBRE, 1956 No. 10

STUDIES ON IMMUNITY TO SCHISTOSOMIASIS MANSONI; EVALUATION OF THE CIRCUMOVAL PRECIPITIN TESTS AS A DIAGNOSTIC PROCEDURE IN CLINICAL SCHISTOSOMIASIS MANSONI; REPORT OF 46 CASES

*RAFAEL RODRIGUEZ-MOLINA**
*JOSE OLIVER-GONZALEZ***
*DIANA G. SERRANO****

Antibodies against the eggs of **S. mansoni** have been detected in the sera from humans infected with this parasite. Precipitins are formed around the membrane when the eggs are incubated in the serum at 37°C. The antibody activity of sera from chronic cases of schistosomiasis mansoni is apparently greater than the active sera from early infections. This enhanced activity in older infections is believed to be a factor of paramount importance in the diagnosis and prognosis of clinical schistosomiasis mansoni. As it is believed that in Puerto Rico the great majority of infections are acquired during childhood or adolescence, the precipitin circumoval reaction might point to the presence of infections when other orthodox methods of diagnosis have failed. Experience has demonstrated that the presence or absence of ova in the feces and rectal biopsy as diagnostic criteria, and following therapy, have yielded equivocal results in many instances. In other words, the criteria now available to determine the effectiveness of a drug in the treatment of schistosomiasis mansoni is inadequate. Thus it is believed that by performing circumoval tests in large group of individuals suffering from this disease, who may or may not have been treated, a dual purpose may be accomplished:—

(1) It will be possible to evaluate the reliability of the circumoval precipitin reaction as a diagnostic test; and (2) the test may be employed as another criterion to determine the results of treatment, along with other criteria currently in use, such as im-

* Clinical Profesor of Medicine at the School of Tropical Medicine, University of Puerto Rico and Assistant Director, Professional Services for Research, Veterans Administration Hospital, San Juan, Puerto Rico; **Professor of Parasitology, School of Medicine, School of Tropical Medicine, University of Puerto Rico; ***Research Technician, San Patricio VA Hospital, San Juan, P. R.

provement in clinical manifestations of the disease and the presence of living and dead ova in the stools and in the small piece of tissue removed from one of the rectal valves.

The present report deals with the evaluation of the circumoval reaction in forty-six proven cases of schistosomiasis mansoni. Living or dead ova were observed either in the stools or in the biopsied rectal tissue at the time when the circumoval test was performed.

MATERIAL AND METHODS

Male Puerto Ricans, United States veterans irrespective of age who are known to harbor infection with S. Mansoni were the subjects for the study. The majority of individuals were cases of schistosomiasis which in the past were hospitalized at San Patricio United States Veterans Administration Hospital at San Juan, Puerto Rico, U. S. A. for disease conditions other than schistosomiasis, the diagnosis of this condition having been established by routine fecal examination; infection with **S. Mansoni** being considered as an incidental finding not related to the primary disease responsible for the hospitalization of the case. A small (not over 10) number of individuals presented clinical manifestations such as abdominal pain associated with bloody stools and tenesmus. Also in the group there were several cases of visceral schistosomiasis, with hepatosplenomegaly, chemical evidence of impaired liver function, associated with anemia, leukopenia and thrombocytopenia. Some cases presented evidence of portal hypertension such as history of repeated hematemesis and demonstrable esophageal varices by x-ray examination.

Eight to ten cubic centimeters of venous blood were withdrawn from cubital veins of arms and allowed to coagulate in order to obtain sera.

The technique of setting up a circumoval test is as follows:

Material Required:

(1) Livers from infected mice or hamsters, (2-4) the animals been infected during 8 to 10 weeks.
(2) Osterizer or electric blendor (washed and cleaned with distilled water).
(3) 1.7% saline at room temperature.
(4) Inactivated sera from infected animals or human subjects (30 minutes at 50ºC; ten minutes if re-inactivity is required.
(5) Two sieves (mesh 50 and 100) to fit test cups, (250 or 500cc).
(6) Five culture tubes for discarding washes.
(7) Glass slides and cover slips.
(8) Vaseline.
(9) Capillary pipettes and rubber balls.

Method:

(1) After crushing a piece of liver between two slides and examining it for ova, homogenize livers in osterizer using 1.7% saline (1 to 2 inches of saline from bottom of osterizer cup); run osterizer for 3 minutes. The 1.7% saline is used to prevent hatching of eggs.

(2) Pass homogenized mixture thru both sieves (coarse on top) rinsing osterizer cup with some more saline into a test cup.

(3) Sediment for 15 minutes at room temperature; meanwhile set 5 tubes in row on a rack.

(4) With a capillary pipette collect sediment from bottom and pour into first tube (collect enough sediment to fill first tube).

(5) Allow sediment in the tube to settle for three minutes and then collect supernatant into second tube.

(6) Add saline again to first tube and allow to settle for another 3 minutes, then collect supernatant into third tube.

(7) Add saline again to first tube and repeat process.

(8) Use sediment in first tube as the antigen for the test (determine concentration first).

(9) Place one drop of serum on the slide, and one drop egg suspension; cover with a vaseline bonder cover slip.

(10) Incubate overnight at 37°C and read test under the microscope.

(11) Observe reaction around living eggs only (hyaline finger-like projections, sometimes septate). Be sure to distinguish true precipitates from artefacts. Count all living ova and those with precipitate around them. Also note intensity of precipitation.

Record Test as Follows:

Name of Patient or animal	Date of Test	Positive Control Serum	Number of Ova on Slide	Number of Positive Ova	Degree of Precipitate	Remarks

Reading and reporting of results: Positive and Negative for circumoval precipitins—(strong, moderate and weak reactions).

Results:

The table shows results of circumoval reaction in the 46 individuals studied. A positive reaction was obtained with the sera of

43 out of the 46 cases, an incidence of positive reaction of 93.4%. Repeated tests were carried out in the three cases in which negative results were obtained and the reactions were again negative. All three individuals giving a negative test were known to have had schistosomiasis for several years and had received repeated courses of Stibophen (Fuadin). Two of them showed dead ova both in stools and in rectal tissue, but the third case had living ova in the stools and in the rectal tissue. No explanation for negative circumoval test is postulated at present.

It is expected that in a near future a larger number of circumoval tests will be performed in a longer series of patients of known infected as well as non-infected controls. Future work on this subject will also include the role of this reaction as another criterion employed to determine results of treatment in human and experimental schistosomiasis mansoni.

CIRCUMOVAL PRECIPITIN REACTIONS IN PUERTO RICAN MALES SUFFERING FROM SCHISTOSOMIASIS MANSONI, TREATED AND UNTREATED WITH STIBOPHEN (FUADIN). LIVING AND OR DEAD OVA IN STOOLS OR IN RECTAL BIOPSIED TISSUE WERE OBSERVED AT TIME WHEN TESTS WERE PERFORMED

Total number of Cases	Positive Reaction Cases — %	Negative Reaction Cases — %
46	43	3
	93.4%	6.5%

SUMMARY

1. The circumoval precipitin test was employed as a diagnostic procedure in clinical schistosomiasis mansoni. The present report is a preliminary study on the reliability of this procedure in the diagnosis of Schistosomiasis Mansoni.
2. The test was carried out in 46 documented cases of S. Mansoni. Living or dead ova were observed in all cases either in the stools or in tissue removed from a rectal valve or in both at the time the precipitin reaction was performed.
3. The circumoval reaction was found to be positive in 43 out of 46 individuals with an incidence of 93%.

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SCHISTOSOMAL HEPATOSPLENOMEGALY

*HECTOR F. RODRIGUEZ, M.D.**

During Dr. Chester M. Jones' visit to Puerto Rico last year, Dr. Jones being Clinical Professor of Medicine at Harvard Medical School, several cases of hepatic bilharzial fibrosis were presented to him. In a letter addressed to me on his return to Boston he stated: "It is really very striking to see the degree of portal hypertension that you experience in your schistosomiasis cases with liver involvement, without the tremendous degree of hepatic insufficiency that we are accustomed to see in cases that we classify in general as cirrhosis. There is certainly a striking difference between the usual cases of chronic liver disease that we see and those that you encounter, that are secondary to this parasitic infestation." The clinical differences of cases with hepatic fibrosis of schistosomal origin and those suffering from portal cirrhosis of alcoholic or nutritional origin have been previously emphasized by us in an earlier report.[1] As this study was limited to male veterans, a similar review of 110 cases of cirrhosis was undertaken at the Ponce District Hospital, where younger patients and of both sexes are hospitalized.

MATERIAL AND RESULTS

Table 1 illustrates the incidence of each etiologic factor in the development of cirrhosis. Sixty (54.5%) patients were chronic alcoholics. Schistosomiasis was found to be the etiologic agent in 33 (30.1%) patients, and a contributing factor in an additional eleven (10.9%) of the cases. These eleven subjects were also alcoholics; therefore, they were excluded from the comparative evaluation. There were five cases associated with other causes, such as biliary system disease and viral hepatitis.

TABLE I

Etiologic factor in 110 cases of cirrhosis

Etiologic factor	No. of Cases	Percentage
Alcoholism and / or malnutrition	60	54.5
Schistosoma mansoni alone	33	30.1
Schistosoma mansoni and alcoholism	12	10.9
Others	5	4.5

* Chief Medical Service, Ponce District Hospital, Ponce, P. R.

TABLE 2

Age and sex distribution of cases with portal and bilharzial cirrhosis

AGE	Portal cirrhosis (60 cases)		Bilharzial cirrhosis (33 cases)	
	No. of cases	Percentage	No. of cases	Percentage
12-19	1	1.7	16	48.5
20-29	3	5.0	9	27.2
30-39	14	23.3	4	12.2
40-49	15	25.0	3	9.1
50-59	9	15.0	1	3.0
60-69	12	20.0	—	—
70-up	6	10.0	—	—
Average age (years)	50.9		23.8	
SEX				
Male	38	63.3	22	67
Female	22	36.7	11	33

Table 2 indicates the age and sex distribution. The cases of bilharzial cirrhosis were found to be much younger, the average age being 23.8 years, younger than the patients with portal cirrhosis by more than twenty five years. It should be noted that in 75% of the schistosomiasis cases, the average age was less than 30 years, while only four (6.7%) patients of the Laennec's cirrhosis group were in that age span. As far as sex, a similar predominance of the male sex was apparent in both groups. The higher incidence of portal cirrhosis in male subjects is explained on a higher number of male alcoholics. Schistosomal hepatosplenomegaly has also been reported more often in the male sex,[2,3] the reason offered being their more frequent exposure to the parasite while river bathing.

The symptoms in both groups are outlined in Table 3. The main difference is the higher incidence of symptoms associated with bleeding varices, melena and hematemesis, in the group with bilharzial hepatosplenomegaly. The presenting symptom of an abdominal mass, in every case an enlarged spleen, was the most frequent complaint in this group, and was rarely encountered in the analysis of cases with portal cirrhosis. On the other hand loss of sexual libido and swelling of the legs and/or abdomen were frequent symptoms in Laennec's cirrhosis, and rare ones in the

TABLE 3

Incidence of symptoms in portal and bilharzial cirrhosis

Symptoms	Portal Cirrhosis (60 cases)		Bilharzial Cirrhosis (33 cases)	
	No. of cases where found	Per cent	No. of cases where found	Per cent
Anorexia	50	83.3%	10	30.3
Malaise & weakness	47	78.3	17	51.5
Swelling of abdomen & or leggs	45	75	3	9.0
Bloating	38	63.3	5	15.1
Abdominal pain	35	58.3	15	45.4
Loss of libide	25	41.6	3	9.0
Fever	22	36.7	7	21.2
Weight loss	18	30.0	4	12.1
Nausea and vomiting	18	30.0	5	15.1
Diarrhea	17	28.3	5	15.1
Melena	16	26.7	14	42.4
Hematemesis	13	21.6	11	33.3
Toxic delirium	11	18.3	1	3.0
Abdominal mass	4	6.7	20	60.6

bilharzial fibrosis group. Other symptoms, such as weakness, anorexia, fever, nausea and vomiting, and abdominal pain showed no marked difference in frequency in the two groups.

The analysis of symptoms indicate the more frequent occurrence of portal hypertension in bilharzial hepatic fibrosis, while the tendency to fluid accumulation and endocrine disturbances were found to be frequent features of Laennec's cirrhosis and rare ones in schistosomiasis.

The relative incidence of physical signs are shown in Table 4. Hepatomegaly was the most frequent physical sign in both groups. Splenomegaly was encountered more often in cases of Schistosoma mansoni cirrhosis (87.9%) than in subjects with portal cirrhosis (35%). As is apparent from this table, the only frequent physical signs in bilharzial cirrhosis are hepatomegaly and splenomegaly. Splenomegaly in schistosomiasis is explained on the basis of mechanical factors, i.e. intrahepatic periportal obstruction, or less frequently, to a toxic factor. As a rule, the splenic enlargement is not associated with any specific lesion, as the presence of ova in the spleen is an exceptional finding.

TABLE 4

Incidence of physical signs

Signs	Portal Cirrhosis		Bilharzial Cirrhosis	
	No. of cases where found	Per cent	No. of cases where found	Per cent
Hepatomegaly	46	76.7	30	90.9
Edema	45	75	4	12.1
Ascites	44	73.3	4	12.1
Loss of body hair	37	61.6	—	—
Cachexia	30	50	5	15.1
Jaundice	29	48.3	2	6.0
Spider nevi	29	48.3	2	6.0
Soft testicle	27	45	—	6.0
Splenomegaly	21	35	29	87.9
Liver palms	20	33	1	3.0
Hyporeflexia	17	28.3	—	—
Gynecomastia	14	23.3	1	3.0
Fetor hepaticus	11	18.3	1	3.0
Coma	11	18.3	1	3.0
Hydrothorax	7	11.6	—	—

The hematologic findings are enumerated in Table 5. As was to be expected, in the bilharzial cirrhosis subjects, where splenomegaly was almost always encountered, hypersplenism was most frequently seen. Thus, leukopenia and thrombocytopenia were found more often in the bilharzia group. Eosinophilia was present in eleven (33%) subjects with bilharzial hepatosplenomegaly and only in five (8.3%) patients with Laennec's cirrhosis.

TABLE 5

Hematologic Findings

Finding	Portal Cirrhosis		Bilharzial Cirrhosis	
	Found in	Percentage	Found in	Percentage
Anemia	30	50	14	42.4
Thrombocytopenia*	5	29.7	15	60
Leukocytosis	16	26.7	3	9
Leukopenia	9	15	14	42.4
Eosinophilia	5	8.3	11	33

* Platelet counts were performed in 17 cases of portal cirrhosis and in 25 of bilharzial cirrhosis.

The liver function tests are presented in Table 6. The results in the portal cirrhosis group showed the expected findings associated with marked hepatic insufficiency, most of the deter-

TABLE 6

Liver Function Tests in Portal and Bilharzial Cirrhosis

TEST	Portal Cirrhosis			Bilharzial Cirrhosis		
	Cases where done	Abnormal in	Percentage	Cases where done	Abnormal in	Percentage
Bromsulphalein	41	36	87.8	29	17	58.6
Thymol turbidity	30	27	76.7	18	9	50
Hanger	51	36	70.6	31	17	54.8
Prothrombin time	42	29	69	24	8	33.3
Alkaline phosphatase	34	20	58.8	17	11	64.7
Van den Bergh	50	29	58	23	4	17.4
Cholesterol total	45	26	57.7	24	13	54.1
less than 150 mg %		22			13	
more than 250 mg %		4				
Hyperglobulinemia	59	25	42.3	30	16	53.3
Inverted A/G ratio	59	24	40.6	30	7	23.3
Albumin less than 3.0 gm. %	59	24	40.6	30	1	3.3
Protein less than 6.0 gm. %	59	17	28.8	30	4	13.3

minations showing a high incidence of abnormality. In bilharzial cirrhosis the liver function tests most often affected were those dealing with protein metabolism (cephalin-cholesterol flocculation, thymol turbidity, serum globulin), in addition to the bromsulphalein dye retention test and the alkaline phosphatase determination. What is most interesting, however, is that a relatively high percentage of cases showed no liver function impairment by laboratory tests, normal function being encountered in 13 (40%) out of 32 patients in which laboratory determinations were performed. In the alcoholic group only three (5%) cases had normal hepatic function tests. The finding of a normal liver profile in subjects with schistosomiasis has also been reported by others[2] and emphasized by us in our communication on splenoportography.[1] Often, one might consider extrahepatic portal obstruction in cases with bilharzial hepatosplenomegaly and unimpaired liver function, especially when, as is not infrequent, the Schistosoma mansoni ova may be absent in the stools or rectal biopsies. It is in these cases that splenic venography is quite valuable in establishing the presence of an intrahepatic block to the portal circulation and ruling out a portal or splenic vein thrombosis. El Ghoulmy et al[2] in evaluating liver affection as judged by the clinical picture, laboratory studies and liver biopsies, reported that the physical examination in patients with schistosomiasis was positive in 40% of the cases, laboratory methods

showed liver function impairment in 24.5% only, while liver biopsy showed definite evidence of bilharziasis in 78.8% of the subjects.

TABLE 7

Incidence of Esophageal Varices

Portal Cirrhosis (60 cases)		Bilharzial Cirrhosis (33 cases)	
No. of Cases Found	Percentage	No. of Cases Found	Percentage
15	25	22	67

Table 7 shows the frequency of esophageal varices in the groups being compared. Fifteen (25%) cases of portal cirrhosis were found to have esophageal varices while in the bilharzial group varices were encountered in 22 (67%). As to the methods used in establishing the presence of varices in bilharzial fibrosis, table 8 indicates the three procedures used and their relative accuracy. Twenty five patients had one or more of these diagnostic studies, varices being found in 17. Of the eight having negative studies, three subsequently had hematemesis and varices were thus diagnosed, two of them dying and one recovering after a shunt operation. Two additional cases had no studies performed but varices were found at autopsy, one dying from cardiac arrest during intraesophageal ligation of bleeding varices and the other from acute cor pulmonale due to pulmonary schistosomiasis. It may be concluded from this that the combination of the three studies is most effective in diagnosing esophageal varices, esophagoscopy having an edge over splenoportography, which seems to be more effective than esophagograms in demonstrating varices. The greater efficacy of splenoportography over barium swallow in showing esophageal varices has been previously reported by Atkinson et al.[5] Figures 1 and 2 depict two cases of bilharzial fibrosis with portal hypertension, in which percutaneous splenic portography gave valuable information as to the status of the portal circulation. The first one clearly demonstrates the presence of large esophageal varices and the increased caliber of both splenic and portal veins. The other one also indicates portal hypertension, intrahepatic in origin, with good visualization of the inferior mesenteric vein, the vessel seen coursing along the left side of the abdomen to the left lower quadrant. These collaterals are not seen in normal subjects and are indicative of stasis of the portal circulation.

TABLE 8

Methods used in the diagnosis of esophageal varices in bilharzial cirrhosis

Method	Done in	Positive	Negative
Barium swallow	20	9	11
Splenoportogram	16	10	6
Esophagoscopy	8	7	1

Causes of Death:

Sixteen (26.7%) patients with portal cirrhosis died, the cause of death being hepatic coma in 13 and shock secondary to bleeding varices in three. In the schistosomiasis group, six (18.1%)



Figure 1: A splenoportography in a case of portal hypertension due to Schistosoma mansoni of the liver. The splenic and portal veins are wider than normal. Large varices are visualized.



Figure 2: The splenic and portal veins are widened and tortuous in their course. The inferior mesenteric vein is seen coursing from the splenic vein along the left side of the abdomen.

died. Three were surgical deaths, one in irreversible shock during an unsuccessful attempt at a splenorenal shunt, one following splenectomy in which thrombocythemia resulted in cerebral thrombosis, and the other in cardiac arrest during emergency intraesophageal ligation of bleeding esophageal varices. The other three cases included one patient with irreversible shock due to bleeding varices, one died in hepatic coma and the third from acute heart failure secondary to pulmonary schistosomiasis. This latter complication is being recognized more frequently at present and has been the subject of an excellent review by Marchand and his group.[6] A brief summary of our case is included to illustrate the features of this rare complication of bilharzia.

CASE SUMMARY

P. V. T., case #58625, a 49 year old man was admitted to the Medical Service of the Ponce District Hospital on August 13, 1956 with severe dyspnea and orthopnea. Examination revealed an acutely ill patient in severe respiratory distress. Despite his marked shortness of breath, the lung fields were not markedly congested. There was a tachycardia and a systolic grade 2 murmur at the apex. Hepatosplenomegaly was encountered. The blood pressure was unobtainable, his temperature was 35°C and the pulse 130 per minute. An electrocardiogram (see figure 3) showed sinus tachycardia, right axis deviation and right ventricular hypertrophy with marked clockwise rotation in the longitudinal axis. P waves were tall and peaked indicating right atrial hypertrophy. Treatment consisted of oxygen, intravenous digitalization and sedation. The patient expired seven hours after admission. The post mortem showed marked cardiac hypertrophy, especially of the right atrium and right ventricle, pulmonary schistosomiasis, "pipe-stem" cirrhosis of the liver, and congestive splenomegaly. Thus, this case illustrates a case of pulmonary schistosomiasis in which the parasite's ova produced embolization of the pulmonary arterioles with increase in pulmonary pressure leading to acute cor pulmonale and death.

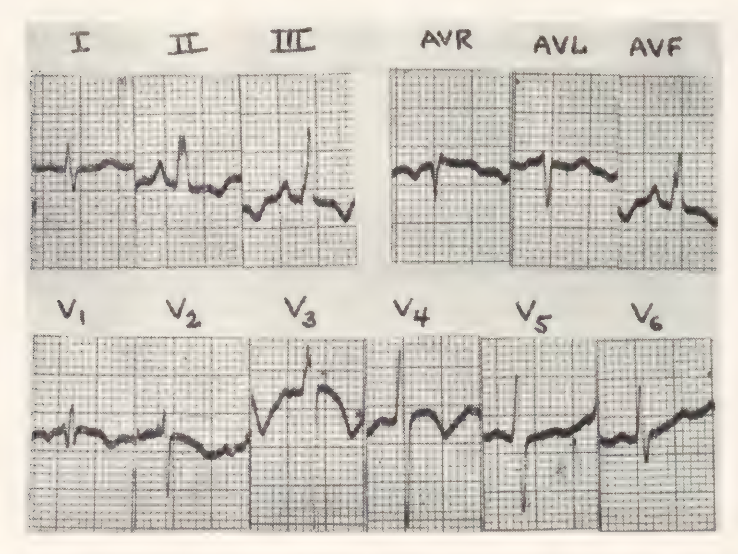


Figure 3: Electrocardiogram showing right axis deviation and right ventricular hypertrophy with marked clockwise rotation in the longitudinal axis. P waves tall and peaked (see leads 2, 3, AVF) indicating right atrial hypertrophy. Case of bilharzial hepatosplenomegaly with pulmonary schistosomiasis leading to acute cor pulmonale and death.

SUMMARY

Liver involvement in schistosomiasis and the exact role that the parasite plays in producing the clinical picture previously described by us is still a controversial subject. Jaffe[7] claimed that the lesions of the liver were due to destruction of the liver cells accompanied by regeneration and increase in connective tissue, thus classifying them as cirrhotic. Karsner[8] opposed this idea and described hepatic bilharzial lesions as periportal fibrosis. More recently, Symmers[9] and Stransky and Pesigan[10] have stated that the condition is due to a nutritional defect and not to the effect of the Schistosoma mansoni ova per se.

Sixteen of our cases have had pathologic examination of the liver, either by liver biopsy in living subjects or by post mortem examination. Of the sixteen cases, pathologic changes were found in thirteen (81.3%), three of the patients having normal needle biopsies. This agrees with the findings of others[11] who have emphasized the evident limitation of examining small pieces of liver tissue in a condition which is mainly focal.

Clinically, bilharzial cirrhosis has been shown to occur in young patients who usually show only hepatosplenomegaly when examined. Portal hypertension and its sequela, esophageal varices, is its outstanding complication. Laboratory studies reveal frequent evidence of hypersplenism and impairment of liver function tests to a moderate degree. In 40% of the cases normal liver function was encountered. The marked hepatocellular damage usually encountered in portal cirrhosis which leads to fluid accumulation, jaundice and signs and symptoms attributed to endocrine disturbances are notoriously absent.

In conclusion it may be said that this study and the previous one have shown that liver involvement by schistosomiasis leads to a definite clinical picture which is characterized by hepatosplenomegaly without severe liver damage and whose outstanding complication is its high incidence of esophageal varices. The management of portal hypertension in these cases lies on a rational approach by surgical groups skilled in the performance of the indicated type shunt operation. The surgical mortality in these cases, because of the relatively good liver function and the youth of the subjects, should be appreciably lower than that of patients with portal cirrhosis of alcoholic or nutritional origin.

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LAS MANIFESTACIONES PULMONARES DE LA ESQUISTO-SOMIASIS DE MANSON EN PUERTO RICO

*E. MARTÍNEZ-RIVERA, M.D.,**
*y ENRIQUE KOPPISCH, M.D.***

INTRODUCCION

Se adquiere esta parasitosis por el contacto de la persona con aguas contaminadas. El huésped intermediario, un caracol, se infesta al penetrar en él el miracidio que sale del huevecillo cuando las excretas humanas se depositan en estas aguas, o cuando son arrastradas hasta ellas por las lluvias.

Luego de transformarse el miracidio, y de multiplicarse en el caracol, salen de éste las cercarias de cola bifurcada, la forma larvaria que penetra en el hombre. La penetración es casi siempre por la piel, pero puede ocurrir por las mucosas. Sólo se introduce la cabeza, desprendiéndose la cola. Desde aquí, hasta que se hace adulto, el parásito se denomina "Metacercaria".

Pasan la piel, penetran los linfáticos, atraviesan los ganglios regionales y alcanzan los grandes troncos linfáticos, abdominal y torácico, llegando así a la sangre venosa y al lado derecho del corazón.[1, 2] Pasan luego al circuito pulmonar a lo largo de los vasos sanguíneos, e irrumpen en el lado izquierdo del corazón. Se supone que sólo puedan subsistir las metacercarias que logran llegar del lado arterial al venoso, siempre en el torrente sanguíneo, en aquellas partes del abdomen cuyo drenaje venoso es a la vena porta.

BASE Y MATERIAL DE ESTUDIO

Hemos llevado a cabo una revisión del material autópsico de la Escuela de Medicina Tropical y Escuela de Medicina, en San Juan de Puerto Rico, limitándonos a un estudio de las lesiones pulmonares, y de su frecuencia a lo largo de los últimos 24 años. Además incluiremos un resumen sucinto de lo más sobresaliente, sobre este aspecto de la enfermedad, que se haya publicado aquí y en otras partes del mundo.

Revisamos 2,000 autopsias consecutivas, practicadas entre el 13 de mayo de 1926 y el 10 de abril de 1950. Al entresacar todos los casos de esquistosomiasis observamos que el más joven

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* Jefe Servicios Médicos, Sanatorio Alejandro Ruiz Soler, Río Piedras, Puerto Rico.

** Profesor de Anatomía Patológica y Jefe de Departamento, en la Escuela de Medicina — Escuela de Medicina Tropical, de la Universidad de Puerto Rico, San Juan, Puerto Rico.

contaba 6½ años de edad. En nuestro país, por lo menos, sería verdaderamente excepcional, y hasta accidental, la infestación antes de que el niño adquiera la edad de 6 a 7 años, que es cuando comienza a tener alguna libertad de acción, y mayor actividad fuera del hogar. Por eso decidimos excluir de la serie todos los menores de 6 años, así como aquellos en que no se examinaron los pulmones microscópicamente, y los extranjeros de corta residencia en este país. El total de los excluídos fué de 480, lo cual dejó 1,520 casos como aceptables para nuestro estudio.

Hallazgos de autopsia:

Entre los 1,520 se encontraron 248 que padecían de esquistosomiasis mansónica, lo cual significa una frecuencia de 16.3 por ciento. No deberá esto de interpretarse como la frecuencia necrópsica para la isla de Puerto Rico, puesto que la mayor parte de los casos de esta serie no procedían de zonas endémicas. Además, éste es un factor casi imposible de analizar por lo frecuente y fácil de los traslados de la zona rural a la metropolitana de San Juan.

TABLA I

DISTRIBUCION DE LOS 248 CASOS DE ESQUISTOSOMIASIS SEGUN LA EDAD, EL SEXO Y LA RAZA

EDAD	SEXO		RAZA				Total	Por Ciento
	Masc.	Fem.	Blancos	Mulatos	Negro	No determinada		
De 6 a 10 años	4	2	6	0	0	0	6	2.42
11-20	27	10	26	9	1	1	37	14.92
21-30	61	22	60	18	5	0	83	33.46
31-40	33	5	25	7	4	2	38	15.32
41-50	34	4	22	12	4	0	38	15.32
51-60	16	4	14	3	3	0	20	8.06
61-70	7	0	6	0	0	1	7	2.81
71-80	1	1	1	0	0	1	2	0.80
No especificado	14	3	8	4	3	2	17	6.85
TOTAL	197	51	168	53	20	7	248	100.00

En segundo lugar, conviene señalar lo siguiente en cuanto al diagnóstico postmórtem. Hemos pasado el diagnóstico en el hallazgo de seudotubérculos con cascarón o huevecillo, o en el hallazgo de cascarones o huevecillos solos, en algunas de las vísceras examinadas microscópicamente. Señalaremos que los órganos más frecuentemente afectados son el recto y el hígado. Del recto se toma siempre un corte a nivel de la válvula de Houston, y del hígado se toman siempre dos cortes de distintas partes. Se comprenderá que la cantidad de tejido examinada está en proporción mínima con el volumen total de esas dos vísceras. Ottolina[3] encontró que por el método de la biopsia rectal la frecuencia puede llegar a un 25 ó 28 por ciento, cuando entre las autopsias practicadas en la misma región, ésta no pasa de un 15 por ciento.

En la Tabla I aparecen los esquistosómicos agrupados de acuerdo con la edad, el sexo y la raza. Resalta de esta tabla que de un 2.4 por ciento entre los 6 y los 10 años, aumenta rápidamente la frecuencia hasta un 33.4 entre los 21 y los 30; luego se mantiene fija en 15 por ciento desde los 31 a los 50, disminuyendo rápidamente de esa edad en adelante.

En cuanto al sexo, se afectan 4 varones por cada hembra.

No pueden basarse conclusiones sobre la raza por no tener datos exactos en cuanto a proporción racial en los focos endémicos, y el punto de origen de la infestación, en la mayor parte de nuestros casos.

Análisis de localizaciones pulmonares:

Entre los 248 bilhárzicos encontramos 161 con lesiones pulmonares, o sea un 64.9 por ciento. En un trabajo anterior, uno de nosotros (E. K.[4]), señaló que la frecuencia con que se afectan los pulmones depende, en parte por los menos, de la gravedad o intensidad de la bilharziosis. Por ejemplo: la frecuencia fué de 4.3 por ciento en las infestaciones mínimas, de 23.8 en las moderadamente fuertes, y de 63.6 por ciento en los parasitismos bilhárzicos graves. En la serie presente, que engloba la anterior, no hemos subdividido los casos de acuerdo con el grado de intensidad de la bilharziosis.

De los 161 casos con lesiones pulmonares, en 158 éstas eran de grado mínimo, sin importancia anatomopatológica o clínica. En ninguno de ellos hubo síntomas que llevaran a sospechar de su presencia, y en sólo uno de ellos se las descubrió macroscópicamente.

En esos casos, desde el punto de vista microscópico, lo usual es encontrar algunos seudotubérculos dispersos, casi nunca más

de uno o dos por corte. Su composición histológica es la ya conocida: Huevecillo completo en el centro, o sólo restos de cubierta ovular sin embrión, rodeados de una o más células gigantes de reacción a un cuerpo extraño, y de algunas células epitelioideas en forma de mosaico. Por la periferia del nodulillo parece un discreto infiltrado de linfocitos y algunas células plasmáticas y eosinófilos. A veces se ve alguna proliferación fibroplástica alrededor de los seudotubérculos, pero nunca hemos visto nodulillos fibrosos, como los que a menudo aparecen en el hígado. Suponemos que lo usual sea la digestión de la cubierta ovular y la regresión total del seudotubérculo. En estas infestaciones mínimas no se forman los angiomatoides de Shaw y Ghareeb.[5]

Puede suceder, en pocos casos, que aparezcan algunas cubiertas ovulares sin reacción inflamatoria en torno suyo, como también, raras veces, puede encontrarse uno que otro verme adulto en alguna arteria pulmonar. Nunca hemos visto alteraciones en los pulmones que pudieran con certeza atribuirse a la fase temprana de la infestación, durante la cual las metacercarias pasan por los pulmones con la sangre.

Queremos señalar que en el caso individual, el hecho de que existe una infestación grave en el hígado o colon no significa que, necesariamente, las lesiones pulmonares tengan que ser igualmente graves.

En toda la serie de 161 casos con alteraciones pulmonares, sólo encontramos 3 en las que éstas fueran de importancia, desde algún punto de vista u otro, a pesar de que en el total de 1,520 individuos incluídos en esta serie, aparecieron 17 de esquistosomiasis grave.

En uno de estos su importancia fué anatomopatológica únicamente, y en los otros dos hubo repercusión clínica.

ANATOMIA PATOLOGICA

A continuación describimos brevemente estos casos:

Autopsia No. 305: C. L., aet. 29, varón, blanco. — Se le diagnosticó bilharziosis 10 años antes de la muerte, cuando mostraba esplenomegalia, y se encontraron huevecillos de Schistosoma Mansoni en las heces fecales y orina (sic.). Mejoró con tártaro emético, y no volvieron a encontrarse huevecillos durante los siguientes 9 años. Sobrevinieron entonces varias hematemesis repetidas y murió exangüe al tercer día.

Hallazgos de autopsia: **Hígado** de 960 gmos. con cirrosis periportal bilhárzica, de tipo Symmers. **Bazo** de 650 gmos., firme, sin corpúsculos de Malpigio visibles, y con algunos infartos anti-

guos, retraídos y parcialmente calcificados. **Esófago:** No pudo encontrarse el punto de origen de la hemorragia. Grandes várices esofágicas se extendían desde un nivel de 5 cms. por debajo del cricoides hasta el cardias. **Estómago e intestino:** Llenos de sangre hasta la acodadura esplénica; sin lesiones visibles. **Recto:** Aparecía rodeado de tejido fibroso denso, pero sin estrechez. **Pulmones:** La consistencia de ambos lóbulos superiores estaba algo aumentada, palpándose en ellos pequeños nodulillos firmes que, al corte, eran blanquecino-grisáceos, dispersos o en pequeños grupos, y de no más de 1 mm. de diámetro. Alguno que otro de ellos se encontraba en otras partes en ambos pulmones. **Sistema Porta:** Sin vermes. La esplénica mostró un gran engrosamiento en su pared. **Examen de excreta:** Huevos de tricocéfalos únicamente.

Examen microscópico: Hígado: Fibrosis periportal con infiltración linfocitaria. Engrosamiento fibroso de la íntima de las arterias. Escasas estructuras calcificadas en espacios porta, imposibles de identificar, pero seguramente eran restos ovulares. **Bazo:** Gran fibrosis de la pulpa, con disminución en el número de folículos. **Venas Porta, mesentérica Superior y esplénica:** Notable esclerosis de la íntima. **Pulmones:** Los nodulillos representaban tubérculos o seudotubérculos con células gigantes de reacción a cuerpo extraño, y rodeado de tejido fibroso infiltrado de linfocitos y eosinófilos. Se encontraron huevecillos calcificados, reconociéndose en algunos de ellos la espícula subterminal y lateral. Estos huevecillos aparecían en torno de los nóbulos, y no dentro de ellos. No se encontraron bacilos ácidorresistentes, y cabe la posibilidad de una tuberculosis, pero no apareció un foco primitivo manifiesto.

Autopsia No. 732: Este caso aparece resumido con bastante detalle en una comunicación anterior (E. K.) por lo que aquí sólo mencionaremos los puntos más sobresalientes.

P. D., aet. 14, hembra, blanca. Caso clásico de esquistosomiasis en foco endémico, con bazo e hígado palpables, anemia moderada, leucopenia (3,800 a 5,150), eosinofilia (11 a 20 por ciento), y huevecillos en heces fecales. A este cuadro se añadió, desde un año antes de la muerte, la disnea de esfuerzo acompañada de dolores en zona esplénica. Se la trató con Fuadín, tres inyecciones en días consecutivos, de 0.5 ccs. cada una, 5 en días alternos, de 3.5 ccs. cada una. A la mitad del tratamiento apareció un poco de fiebre (100°F) y notáronse sonidos respiratorios ásperos en el vértice derecho. A los 3 días el pulso, que había promediado 104 por min., aumentó a 120, y las respiraciones, de 26 a 30. Apareció una zona de submatidez desde la mitad de la escápula derecha hasta la base pulmonar, acompañada de algunos finos estertores ins-

piratorios. Persistió la tos durante los 17 días de permanencia en el hospital, y a veces la expectoración estuvo teñida de sangre. A los 12 días de comenzar el tratamiento se administró la séptima inyección, a una hora que desconocemos. A las 6 de la tarde la tos se recrudeció, la enferma se negó a comer, y vomitó varias veces. A las 2 de la madrugada hubo una aparente mejoría, pero a las 5:30 sobrevinieron disnea, cianosis y pérdida del conocimiento, falleciendo a las 6:05 A. M.

Examen Macroscópico: Hígado: 870 gmos. Cirrosis periportal moderada, con fibrosis periportal, en anillos y de tipo Symmers. **Bazo:** 360 gmos.; pulpa blanda y pizarrosa. Folículos muy prominentes. **Pulmones:** Derecho 320, e izquierdo, 270 gmos. Se palparon pequeños nodulillos, sobre todo en lóbulos inferiores. Por debajo de la pleura viéronse algunas zonas firmes, violáceas, de unos 0.8 cm. de diámetro, y pequeños focos amarillentos con diámetro de unos 0.2 cm.; estos últimos sobresalían ligeramente sobre la superficie pleural, y a veces estaban rodeados de una estrecha zona congestiva o hemorrágica. Al corte resaltaba la intensa congestión del tejido pulmonar, el cual además aparecía sembrado de nodulillos grises, como de 1 mm. de diámetro, que a veces aparecían en pequeños grupos. Se veían también algunos pequeños focos amarillentos, y pequeñas zonas hemorrágicas de consolidación. Las zonas violáceas de la superficie, al corte parecían infartos recientes. Los ganglios hiliares eran relativamente grandes. Un ganglio situado en la bifurcación de la tráquea tenía un diámetro de 5 cms. En todos ellos aparecían muy prominentes los folículos linfoides, hasta el extremo de que en un principio se interpretó el cuadro ganglio-pulmonar como de tuberculosis miliar. **Corazón:** Pesó 170 gmos., lo cual está en los límites, normales, según la edad del sujeto, pero en relación con las otras vísceras y el cuerpo, parecía algo hipertrófico. El lado derecho estaba ligeramente dilatado e hipertrofiado, siendo el grosor de la pared ventricular de 0.6 cm. en ese lado, y de 1 cm. en el izquierdo. **Tracto gastrointestinal:** Normal, excepto por la gran prominencia del tejido linfático en el intestino delgado y colon. En las porciones distales del recto aparecieron 3 pólipos, dos de ellos sesiles, y el otro pediculado. El mayor tenía un diámetro de 0.5 cm.

Examen Microscópico: Hígado: Abundante pigmento bilhárzico en células de Kupffer. Fibrosis portal moderada, con gran infiltración eosinofílica y, en menor grado, de monocitos y linfocitos. Huevecillos numerosos en algunos espacios porta, escasos en otros, a veces rodeados de una zona de necrosis, y otras veces con formación de seudotubérculos. En algunas partes el tejido fibroso

rodeaba pequeños seudolobulillos hepáticos. **Bazo:** Muy abundante pigmento bilhárzico por toda la pulpa roja, a veces en grumos. Gran prominencia de los folículos. **Pulmones:** Numerosos seudotubérculos con huevecillos en el centro. Algunos eran más viejos que otros; y estaban rodeados de una cápsula fibrosa. Se asentaban mayormente alrededor de bronquios y arterias de mediano calibre, pero algunos se situaban en las paredes alveolares. Se encontraron pequeñas dilataciones aneurismáticas en estas arterias, con gran engrosamiento fibroso de la íntima. En torno de estas arterias se disponían pequeños vasos neoformados, constituyendo angiomatoides. En algunas de esas lesiones se encontraban seudo tubérculos, a veces en la propia pared arterial, otras veces en el tejido adyacente, ya aislados o en grupos. Las arterias de pequeño y mediano calibre mostraban reduplicación de la lámina elástica interna, hipertrofia de la media y endarteritis obliterante.

No es fácil, ni aún en cortes en serie, el trazar con exactitud el comienzo de la formación de los angiomatoides, porque amén de que éstos aparecen en distintos tiempos, se complican por el desarrollo simultáneo de seudotubérculos. El huevecillo llega al pulmón como una embolia, alojándose en pequeñas ramas arteriales. Esto estimula a su alrededor el comienzo del seudotubérculo; al mismo tiempo, o muy poco después, comienzan a aparecer vasos jóvenes de tipo capilar, formándose así los nodulillos que Shaw y Ghareeb[7] llamaron angiomatoides. En los casos graves, como el que se describe, pueden encontrarse tres o cuatro angiomatoides en campos de menor aumento (100 diámetros).

Los pequeños focos amarillentos eran grupos de alvéolos ocupados por fibrina y eosinófilos. En las zonas violáceas se encontraron numerosos hematíes, además de las células mencionadas, y de la fibrina.

Autopsia No. 1385: D. Ll., aet. 29 años, varón, negro.

Dos semanas antes del ingreso experimentó dolor epigástrico intenso y desvanecimiento, por lo cual permaneció varios días en cama. Tuvo entonces diarrea, que tres días más tarde se tornó sanguinolenta. Se pensó en reumatismo poliarticular agudo, pero no existían antecedentes personales. Tampoco había edema de extremidades, si bien dijo que cuando trabajaba mucho le sobrevenía disnea, se acostaba por un rato, y luego reanudaba su trabajo.

Exploración: Temp. 37°C. Pulso 70. Resp. 20. P. A. 120/90.

Individuo bien desarrollado y bien nutrido que no parecía estar enfermo. Edema moderado del dorso de los pies y piernas. El corazón estaba aumentado de tamaño y se oía un soplo sistólico por todo el precordio. La pared abdominal estaba tensa. El hígado pareció aumentado a la percusión, y el tacto de esa región era do-

loroso. No se palpó el bazo. No se llegaron a practicar exámenes de laboratorio. A las 9 horas y media después del ingreso sufrió un fuerte ataque de disnea, y cayó en colapso circulatorio, con piel fría y sudorosa, muriendo en pocos minutos.

Después de la muerte se recogieron datos anamnésicos adicionales. Había sido un hombre saludable. Un año antes perdió el conocimiento después de una corta carrera; sintió dolor precordial y le llevaron a su casa; se sintió bien tras de un descanso de pocas horas. Hace cinco meses sus amigos y familiares notaron que había aumentado en peso. Las diarreas con deposiciones sanguinolentas comenzaron tres días antes de su ingreso en el hospital Se bañaba diariamente en una quebrada que se sabe está fuertemente infestada con el esquistosoma de Manson.

Examen Macroscópico: Cadáver de negro joven, fornido, con ligero edema de ambos tobillos. El vientre no estaba distendido. La cavidad peritoneal contenía unos 1,000 ccs. de líquido transparente y ligeramente amarillento. El borde anterior hepático se encontraba a 2.5 cms. por encima del reborde costal, en la línea medioclavicular derecha. No se visualizaba el bazo. En cada cavidad pleural se encontraron de 200 a 300 cc. de líquido amarillento y transparente. El saco pericárdico contenía 200 cc. de líquido similar. **Corazón:** Pesó 460 gmos., y la hipertropia era del lado derecho. El grosor del ventrículo derecho era de 0.9 cm. y el del izquierdo, de 1 cm. Se vieron algunas petequias por debajo del epicardio sobre la aurícula y ventrículo derecho. La aurícula derecha estaba muy dilatada, su endocardio estaba ligeramente engrosado. Las columnas carnosas de esta misma aurícula aparecían muy hipertrofiadas. El borde libre de la tricúspide se había engrosado ligeramente. El ventrículo derecho mostraba gran dilatación e hipertrofia de los músculos papilares y columnas carnosas. Las demás válvulas, la aurícula y ventrículo izquierdos, y las arterias coronarias eran normales. **Pulmón izquierdo:** Pesó 430 gms. En el vértice se encontró una zona de engrosamiento pleural de no más de 1 cm. de diámetro. Al corte el tejido pulmonar era rojizo, y aparecía sembrado de numerosos nodulillos blanquecinos, ligeramente elevados, con diámetro de no más de 1 mm., distribuídos igualmente por ambos lóbulos. Las arterias pulmonares principales mostraban paredes prominentes; en algunas de las más pequeñas la luz parecía reducida en su calibre. En el lóbulo inferior se encontraron unas zonas induradas como de 1 cm. de diámetro. En el margen superior del lóbulo inferior se encontró una zona cuadrangular y firme, de periferia bien definida y de color rojo oscuro, con un largo de 2.5 cms., y que penetraba en el pulmón hasta 1.5 cms. La mucosa bronquial estaba muy congestionada. **Pulmón**

Derecho: Pesó 490 gmos., y en todo era parecido al izquierdo. **Bazo:** Pesó 290 gmos. y midió 13 x 9 x 3.8 cms. La cápsula era lisa y brillante, excepto en el polo posterior, el cual mostraba algunas adherencias fibrosas. La consistencia era normal. Al corte la pulpa era rojiza, con corpúsculos esplénicos mal definidos y con trabéculas algo prominentes. **Páncreas:** Al corte los lobulillos se dibujaban con mayor prominencia de lo normal. El tejido adiposo peripancreático mostraba múltiples zonas de color anaranjado brillante. **Hígado:** Pesó 1040 gmos. y midió 21.5 x 17.5 x 6 cms. La cápsula era de aspecto blanquecino y de grosor ligeramente aumentado. La superficie del órgano era difusa y finamente granular. Al corte las ramas principales de la porta estaban rodeadas de gruesos collarines blancos y fibrosos desde los cuales se extendían mallas fibrosas finas por entre el parénquima hepático, subdividiéndole en seudolobulillos. **Vesícula Biliar:** La pared era edematosa. Veíanse petequias por la serosa y una fibrosis subserosa difusa. La mucosa parecía normal. **Esófago:** Sin várices esofágicas. **Intestino Delgado, Apéndice e Intestino Grueso:** Normales, excepto por congestión de la mucosa.

Examen Microscópico: **Corazón:** En un corte de ventrículo izquierdo encontráronse dos o tres fragmentos de cubiertas ovulares en el centro de un nodulillo fibroso situado por debajo del epicardio. El ventrículo derecho mostraba hipertrofia de fibras miocárdicas y edema del estroma. **Pulmones:** Las alteraciones fueron en todo similares a las descritas en la **Autopsia 732.** **Bazo:** Gran congestión de la pulpa. Se encontró abundante pigmento pardo oscuro en células reticuloendoteliales. **Hígado:** Gran fibrosis portal. En los espacios porta veíanse numerosos fagocitos con pigmento idéntico al del bazo el cual también formaba pequeños grupos dentro de las células de Kupffer. Estas últimas estaban aumentadas de tamaño por todo el órgano. Se veían huevecillos bilhárzicos ocasionalmente por el tejido fibroso, y algunos de ellos contenían un embrión bien conservado. En el tejido fibroso se veían numerosos vasos capilares y zonas de densa infiltración linfocitaria. En torno de los espacios porta aparecían numerosos seudolobulillos hepáticos. En relación con el número de huevecillos y cubiertas ovulares, viéronse pocos seudotubérculos. También era poco notable la proliferación de conductos biliares. **Vesícula:** Engrosamiento subseroso por edema y fibrosis, sin huevecillos ni seudotubérculos bilhárzicos. **Riñón:** En un corte encontróse en la corteza un huevecillo esquistosómico rodeado de células epiteliodeas que constituían un seudotubérculo cuya periferia estaba infiltrada de eosinófilos y linfocitos. **Esófago:** Los tejidos subepiteliales estaban congestionados, pero no se vieron várices eso-

fágicas. **Estómago:** Apareció un sólo seudotubérculo en las profundidades de la mucosa. **Intestino Delgado:** Se vieron numerosos huevecillos y cubiertas ovulares en la mucosa y submucosa, tanto libres en los tejidos como en el centro de los seudotubérculos. Alrededor de esas estructuras ovulares y de los seudotubérculos disponíanse numerosos eosinófilos. **Apéndice, Colon y Recto:** Similares al intestino delgado. La submucosa mostraba moderada fibrosis.

COMENTARIOS

Desde el punto de vista anatómico, los casos de esquistosomiasis pulmonar grave son de gran interés. Estos se caracterizan por embolizaciones múltiples de huevecillos que obstruyen pequeñas arteriolas pulmonares. El tamaño de estas últimas está determinado por el del huevecillo, el cual en la especie mansónica es ovoide, con longitud de 150 y anchura de 50 micras. La formación de seudo tubérculos en la adventicia arterial, y la proliferación de capilares da lugar al desarrollo de nodulillos que se conocen como "angiomatoides."

En las infestaciones graves, cuando se repiten las embolias de huevecillos en los pulmones, se complican las lesiones arriba descritas con un engrosamiento escleroso difuso de la íntima de las arterias y arteriolas pulmonares. Esto debe de resultar de un aumento de la tensión sanguínea en el circuito pulmonar, y se traduce a su vez en hipertrofia y dilatación del lado derecho del corazón.

Los primeros en hacer un minucioso estudio de las formas pulmonares graves de la bilharziosis fueron Shaw y Ghareeb.[5] En 282 autopsias de casos de esquistosomiasis en Egipto, encontraron lesiones pulmonares debidas a huevecillos en un 33 por ciento. Estos autores escribieron en detalle la histopatología y patogénesis de las lesiones. La complicación con esclerosis de la íntima apareció en un 2.1 por ciento de todos los esquistosómicos y en un 6.3 por ciento de los que tuvieron lesiones pulmonares bilhárzicas. Se encontraron vermes en el pulmón en un 10.5 por ciento de los casos pulmonares.

Desde antes del detallado estudio anatomopatológico de Shaw y Ghareeb[5], Bey[6] en Egipto, Clark Graef[7] en una puertorriqueña nuestra en Nueva York, y Silveira[8] en Brazil, habían ya llamado la atención sobre la endarteritis pulmonar de origen bilhárzico con repercusión en el lado derecho del corazón.

En nuestro país Pons,[9] en un importante estudio clínico de la bilharziosis, se refirió a un caso avanzado de la forma hepatoesplénica que murió con signos de decompensación del lado derecho del corazón, y a otro que tuvo algunos signos de esto último, pero en

ninguno de los dos se practicó la autopsia. El autor se refirió también al segundo de los casos que aquí informamos.

Suárez y Hernández-Morales[10] hicieron en 1944 un análisis de la literatura médica y estudiaron en detalle cinco casos clínicos, todos con manifestaciones pulmonares. El último de éstos, sin embargo, era un asmático, y lo incluyeron los autores precisamente por no creer que el asma fuera de origen bilhárzico.

Los otros cuatro casos eran niños de 12, 11, 15 y 15 años respectivamente. Las manifestaciones clínicas más significativas de afección cardiopulmonar fueron: Cianosis de los dedos, a veces acompañada de dedos hipocráticos, exageración del segundo tono pulmonar, y de ciertas alteraciones radiográficas en los pulmones, tales como fibrosis peribronquial y, en uno de ellos, sombras de infiltración del parénquima, las cuales desaparecieron al tratarse al niño con fuadín.

Desde el punto de vista radiográfico las manifesatciones pulmonares han sido ampliamente estudiadas por Mainzer.[11, 12, 13] Las citas mencionadas no comprenden toda la bibliografía de este autor.

En lo que atañe a estas imágenes radiográficas de los pulmones descritas por Mainzer y otros autores, en Puerto Rico existe un hecho interesante que aún permanece inédito y que consideramos relatar ahora: Por comunicaciones individuales hechas personalmente a uno de nosotros (E. M.), tres de nuestros radiólogos más distinguidos, los doctores M. Guzmán-Rodríguez,[14] J. N. Gándara,[15] y C. Guzmán-Acosta[16] nos informan haber estudiado más de un millón y medio de placas radiográficas del tórax pertenecientes a personas de ambos sexos, abarcando todos los grupos de edades desde la infancia a la senectud, las razas que conviven en nuestro país y las distintas clases sociales de nuestra isla, razón por lo cual se puede decir que el grupo de placas estudiadas es representativo de nuestra población. Añaden ellos: "aunque entre los examinados existe un número considerable de casos comprobados, clínicamente, de infestación por esquistosoma mansoni, no hemos encontrado una sola placa en que la imagen radiográfica sea patognomónica de esta enfermedad, ni siquiera hallazgos radiográficos que sirvan para establecer, con certeza, un diagnóstico definitivo de bilharziosis."

Es un hecho indubitable que, en ciertas áreas endémicas de esquistosomiasis mansoni, con frecuencia acontecen manifestaciones pulmonares que producen lesiones de carácter crónico en los pulmones y corazón. Es igualmente cierto que, en la clínica, el descubrimiento de estas manifestaciones pulmonares va en aumento con el transcurso de los años, gracias a mayores y mejores facilidades

para su diagnóstico, a la sagacidad en los médicos y a la creciente importancia que se le va adjudicando a esta forma de la enfermedad por las autoridades en la materia.

Todos los autores están contestes en que esta condición es capaz de simular las manifestaciones clínicas que suelen encontrarse en las enfermedades congénitas del corazón, la fiebre reumática, la tuberculosis miliar y la pulmonar, las enfermedades congestivas del corazón, el asma bronquial y otras nosogenias.

Es fácil comprobar que las repercusiones pulmonares que, a veces ocurren en el curso de infestaciones por esquistosoma mansoni han sido extensamente estudiadas, desde hace muchos años, por numerosos autores en diversas partes del mundo. Los hallazgos de estos estudios han sido publicados, por los investigadores, en revistas médicas que circulan con profusión. No obstante lo anterior, hasta fecha reciente, la mayoría de autoridades médicas no le habían dado a este asunto la importancia que, a nuestro juicio, merece el tema. Hace muy poco tiempo se le ha otorgado, parcialmente, el valor que esta entidad nosológica puede tener especialmente en los trópicos.

Casi la totalidad de los libros de texto sobre medicina interna al tratar el tema de las infestaciones por esquistosoma a penas si mencionan las repercusiones pulmonares más allá de decir que a veces ocurren. Más sorprendente aún es el hecho del silencio que sobre el mismo tema mantienen los libros de texto de patología general, de enfermedades tropicales y de enfermedades del tórax, publicaciones donde, a lo sumo, se le dedican muy escasas líneas. El libro de Marshall y Perry,[17] "Diseases of the Chest" es uno de los pocos que dedica un capítulo completo a las manifestaciones cardiopulmonares en las esquistosomiasis.

Entre las contribuciones a la histopatología merece mención especial la importante monografía de Lopes de Faria,[18] que apareció en el 1952.

A los interesados en este tema podemos recomendarle, además, los estudios de Alves Meira[19,20,21] Baldo y colaboradores,[22] Bedford,[23] Da Silva,[24] Effan y colaboradores,[25,26] Sirry,[27] Kenaway,[28] Erfan,[29] y Mainzer,[30] y Viswanthan.[31]

RESUMEN

Se traza la ruta de entrada y emigración de las metacercarias hasta su localización en hígado y la oviposición en pared colónica. Las localizaciones pulmonares de vermes y huevecillos podrían obedecer a su paso ocasional, desde ramas del plexo hemorroidal, a la vena cava inferior, llegando así a las arterias pulmonares como embolias.

Revisamos 2,000 autopsias consecutivas practicadas entre los años de 1926 y 1950 en la Escuela de Medicina-Escuela de Medicina Tropical, en San Juan de Puerto Rico.

Descartáronse 480 casos de menores de 6 años —entre los cuales es prácticamente imposible que pueda acontecer esta enfermedad aquí— también extranjeros de corta residencia en el país y autopsias en que no se examinaron los pulmones microscópicamente.

Entre 1520 autopsias restantes se encontraron 248 casos de bilharziosis, con una frecuencia general de un 16.3 por ciento.

Se basó el diagnóstico en el hallazgo de huevecillos, ya sólos, o en el centro de seudotubérculos, en cortes microscópicos de una o más vísceras.

Fluctuó la edad de los afectados entre los 6½ años y los 70. La frecuencia fué de 2.4 por ciento en los de 6 a 10 años, de 33.1 en los de 21 a 30, y de un 15 desde los 31 a los 50, disminuyendo rápidamente de ahí en adelante.

Entre los bilhárzicos hubo 4 varones por cada hembra.

En los 248 bilharziosos se encontraron 161 (64.9 por ciento) con lesiones pulmonares. De éstos últimos, en 158 los pulmones se afectaron en grado mínimo, con escasos seudotubérculos, sin el examen microscópico.

En sólo 3 casos revistieron importancia clínico patológica las lesiones pulmonares.

Se describen, brevemente los únicos tres casos con alteraciones pulmonares de importancia clínica o anatomopatológica, y se presenta un abstracto sucinto de los hallazgos de las autopsias practicadas en estos tres casos.

Se ilustra el trabajo con unas pocas microfotografías interesantes, tomadas de los cortes histopatológicos de los pulmones de estos tres casos.

Entre los 248 bilhárzicos encontramos sólo 17 casos graves o avanzados. Esto representa el 6.9 por ciento de los parasitados y el 1.1 por ciento de la serie total de 1,520 autopsias. Los tres casos de afección pulmonar representan el 1.2 por ciento de los bilharziosos y el 0.2 de la serie total de autopsiados.

En el cuadro global de la esquistosomiasis mansónica en Puerto Rico las manifestaciones pulmonares son de gran interés desde el punto de vista anatomopatológico, aunque de escaso valor relativo en el aspecto clínico. Las lesiones pulmonares se caracterizan por embolizaciones múltiples producidas por los huevecillos que van, arrastrados por el torrente sanguíneo, a los pequeños vasos y obstruyen pequeñas arteriolas pulmonares.

Las manifestaciones pulmonares pueden ocurrir en cualquie-

ra o varias de las diversas etapas evolutivas de la infestación parasitaria, de la que sólo representan una fase particular en una localización determinada.

Como causa de morbilidad y letalidad en Puerto Rico, las manifestaciones pulmonares de la bilharziosis son de escaso interés para el médico práctico, y aun para el especialista del tórax.

Es lo más probable que en los focos endémicos de Puerto Rico, y de otras partes del mundo, las formas pulmonares revistan mayor importancia.

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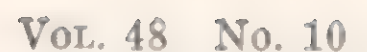
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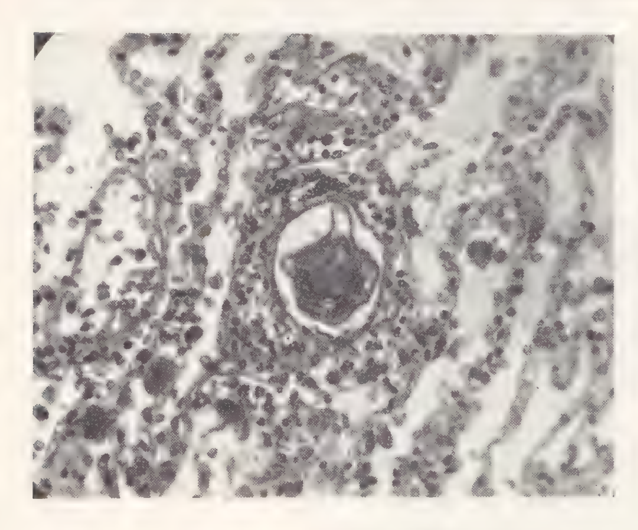


Fig. 1. Huevecillo con embrión; escasa infiltración con linfocitos y algunos eosinófilos. No ha habido tiempo para la formación del seudotubérculo. (X 360)

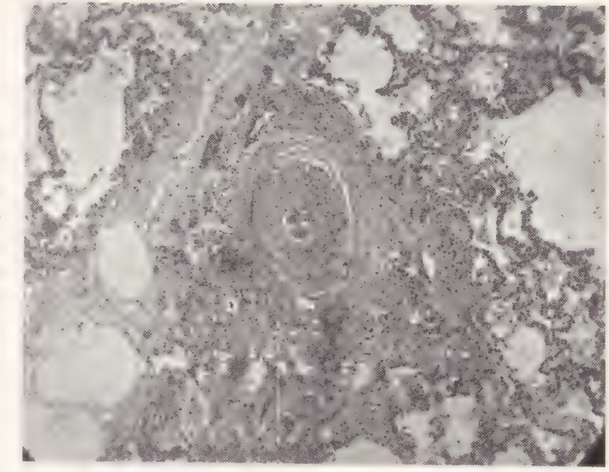


Fig. 2. Seudotubérculo típico; huevecillo en el centro rodeado de gran célula gigante de reacción a cuerpo extraño; zona de células epitelioideas y cápsula fibrosa periférica. (80X)

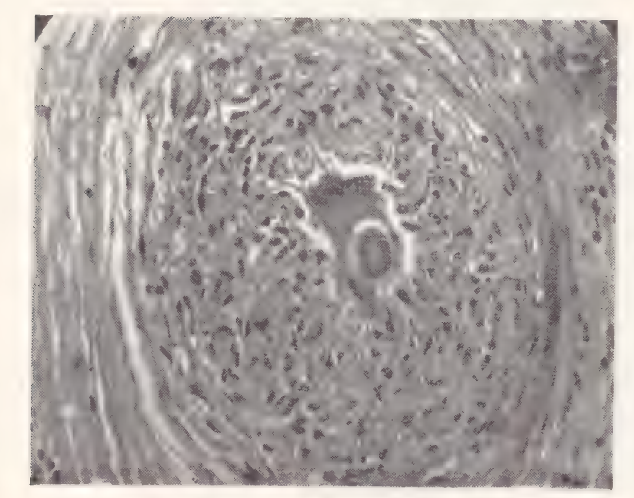


Fig. 3. Mayor aumento de la anterior. (360X)

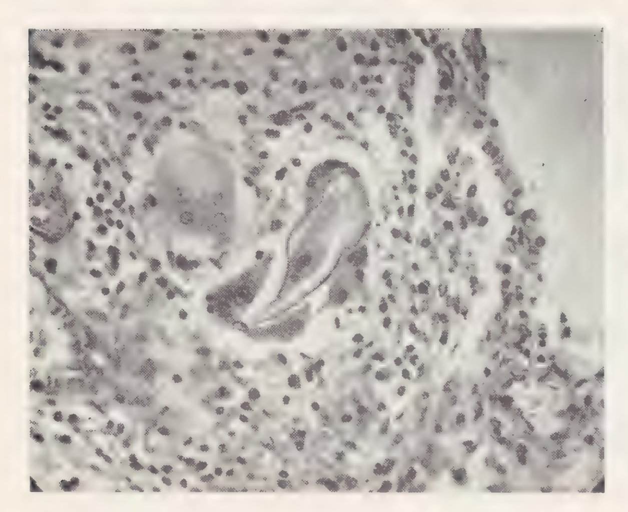


Fig. 4. Cascarón en vías de disolución por células gigantes.

(360 X)

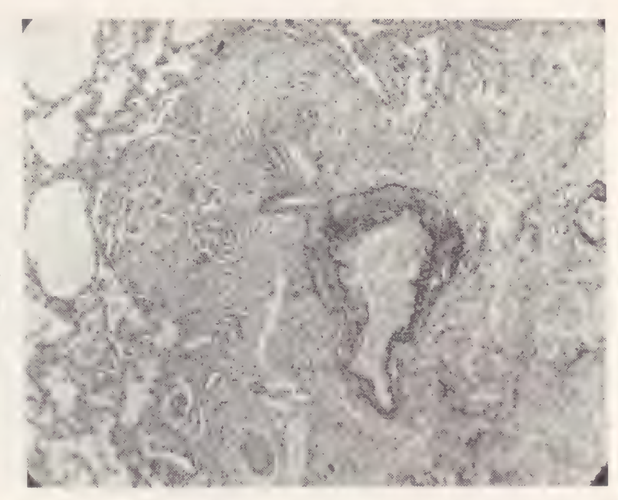


Fig. 5. Reduplicación de fibras elásticas y engrosamiento de la íntima. Capilares neoformados y seudotubérculos en relación con las arteriolas embolizadas. (80 X)

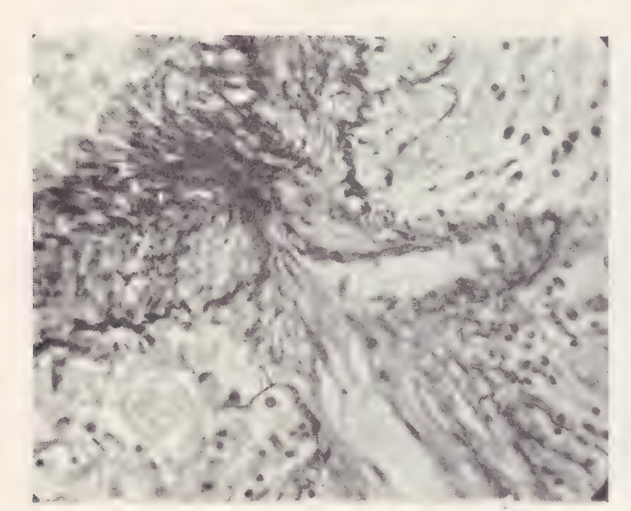


Fig. 6. Detalle de las arteriolas afectadas en el corte anterior. Aumento de fibras elásticas en la arteria de origen. (360 X)

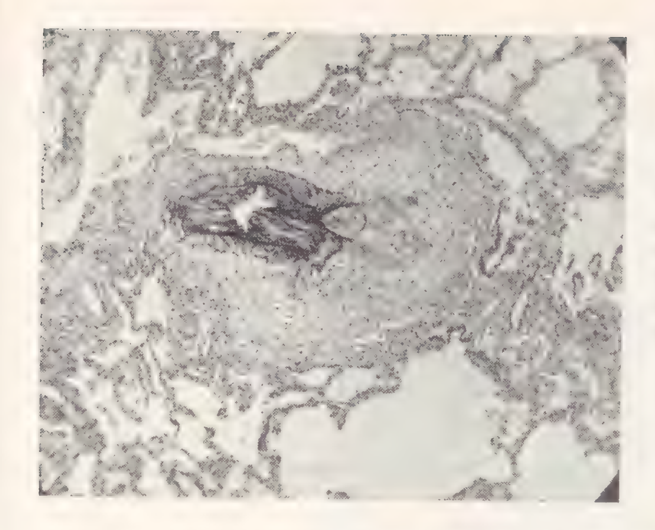


Fig. 7. Gran engrosamiento de la íntima y reduplicación de fibras elásticas en la zona de origen de un angiomatoide. (80 X)

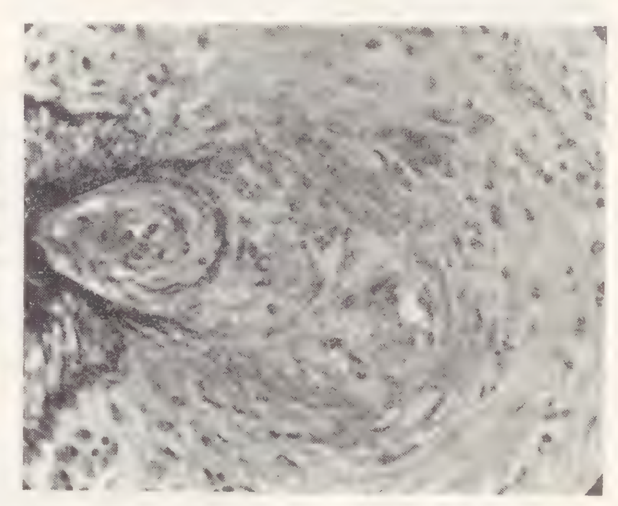


Fig. 8. Detalle de la anterior mostrando el sector de comienzo de una arteriola embolizada; no incluye el huevecillo, pero sí parte de un angiomatoide y seudotubérculo. (360 X)

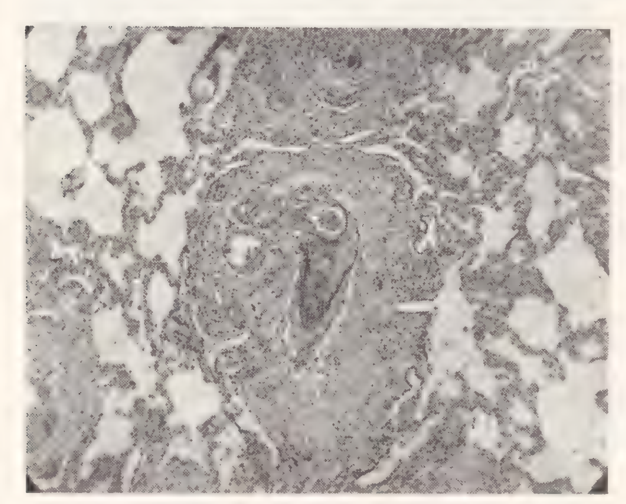


Fig. 9. Angiomatoide con detalles similares a los ya mencionados.

(80 X)

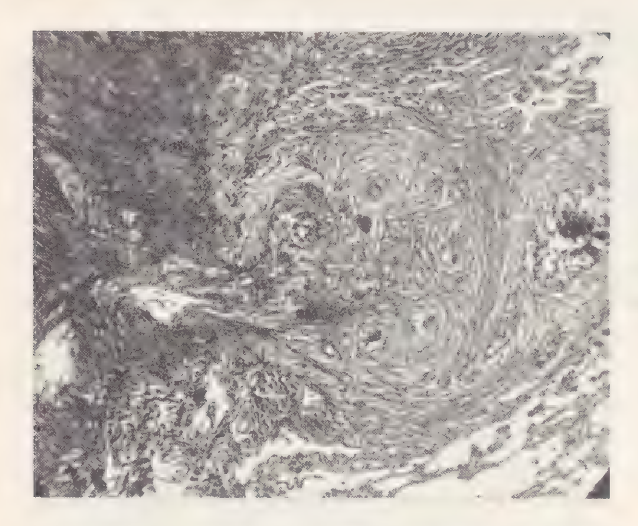


Fig. 10. Angiomatoide y seudotubérculos múltiples formando un nodulillo en relación con arteriolas embolizadas. (80 X)

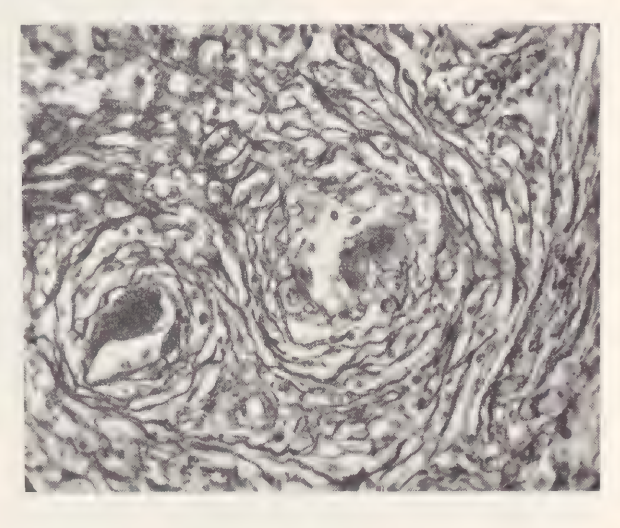


Fig. 11. Detalle del retículo en seudotubérculos. (360)

MANAGEMENT OF ABNORMAL BLEEDING AT TERM*

*WILLIAM F. FINN, M.D.***

I welcome this opportunity to speak to the members of the Puerto Rican Medical Assembly on this practical aspect of Obstetrics. Visits to various hospitals in the last several days have impressed upon me the frequency and the importance of this complication of pregnancy in your country. The following material has been organized not in the classical text book fashion with references but rather from a practical viewpoint, in a chronological order as the problems arise.

CAUSES OF BLEEDING AT TERM

The following table shows the chief causes of bleeding at term.

TABLE I
Causes of Term Bleeding

1. Placental (Major)
   a. Placenta previa
   b. Premature separation of placenta
   c. Rupture of marginal sinus
   d. Vasa Previa
2. Cervical (Minor)

The above table lists the various causes which we will consider. Placenta previa is perhaps the commonest complication and is very simply divided into percentage of the completely dilated cervical os which is covered by placenta so that we have total placenta previa when the entire cervical os is covered; partial placenta previa when any segment of the cervix is so covered; and lastly, low implantation of the placenta when the placental edge impinges upon the edge of the dilated cervix, but does not cover it in any way. We have gradually abandoned the terms central placenta previa and lateral placenta previa, because they were not descriptive enough. Premature separation of the placenta consists of two major types: 1. A mild degree in which bleeding is moderate, tonus of the uterus is increased slightly, fetal heart is still present. 2. A very severe premature separation in which

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** Assistant Professor, Obstetrics & Gynecology, Cornell Medical College; Associate Attending Obstetrician & Gynecologist, New York Hospital; Director, Obstetrics & Gynecology, North Shore Hospital.

there is board-like rigidity of the uterus, marked external bleeding and loss of the fetal heart. Rupture of the marginal sinus is a new concept based on the new knowledge of the anatomy of the placenta. As we well know there is a circular sinus which surrounds the entire placenta and acts as a drainage vein for blood for the placenta. Certain placentas on careful examination show the following characteristics: a) an organized clot at the margin of the placenta; b) a vascular sinus which has been torn; and lastly, c) an organized linear thrombus in the torn sinus. These three characteristics which can be recognized by removing clots from the margins of the placenta constitutes the diagnosis of ruptured marginal sinus. Sometimes symptoms are absent; sometimes they may be severe. The last major cause of bleeding is the so-called fetal bleeding from a torn vein—vasa previa. This is somewhat comparable to rupture of the marginal sinus except that organization and thrombus formation does not occur. Since this bleeding is largely from the fetus there may be marked signs of fetal distress or total absence of the fetal heart. Its immediate recognition and treatment is imperative.

The minor cervical reasons for bleeding at term include such things as cervicitis, endo- and exocervical polyps; cervical erosions; various plaques of decidua on the cervix and carcinoma of the cervix. Distinctions between these can be readily made by smear and biopsy.

MANAGEMENT OF BLEEDING AT ONSET IN HOME

This consists of three parts:

1. What the patient should do.
2. What the doctor should do.
3. Transportation to the hospital.

Patients should be instructed to notify their doctor at the onset of any bleeding. He then can interpret in the light of his knowledge of the patient. The patient and her family should avoid panic and should call the doctor immediately. The doctor on his part, should see the patient, should refrain from performing any internal examination inasmuch as this may cause more bleeding particularly in placenta previa. This internal examination should be deferred until the patient is in the hospital where all facilities for transfusion are available. The pulse and blood pressure should be checked. The amount of bleeding should be appraised. If this is sufficient to warrant hospital admission, the doctor should alert the hospital so that when the patient arrives at the hospital the admission will be facilitated without delay and the patient can be promptly sent to the Delivery Floor where nurses, labora-

tory technicians and other doctors are available to help with the management of this grave emergency. The doctor should then help to arrange the transportation to the hospital and should, if necessary during this trip, administer plasma, serum albumin or a plasma expander such as dextran as a temporary supportive measure if the patient shows signs of shock.

IMMEDIATE MANAGEMENT IN HOSPITAL

As soon as the patient has been admitted to the hospital her history should be re-evaluated. Certain points should be stressed:

1. The amount of bleeding.
2. Whether there have been any previous episodes of bleeding.
3. Whether there is any pain with this bleeding or with other episodes of bleeding.

Painless bleeding suggests placenta previa; episodes of pain suggest certain degrees of separation of the placenta. A careful physical examination is done. The general appearance of the patient is noted. Signs of shock, such as pallor, moisture of skin, low blood pressure and fast pulse are checked. Respirations are noted. The amount of bleeding in the vagina, about the vulva, on the legs and on the clothes is noted. The uterus is felt to see whether it is rigid, whether it is contracting or whether there are tender or painful areas in any part. The fetal heart is listened for; its presence and rate are determined. Presentation of the baby and the station of the presenting part are determined. The clot is inspected to see if it is a firm clot which retracts well and does not crumble on touch. The following laboratory procedures are immediately done:

1. The hematocrit and hemoglobin are determined. Apparently in Puerto Rico many patients have extremely low hemoglobins which they tolerate better than the women in North America who are not so prone to show a chronic anemia.

2. Blood should be typed, cross matched and should be available for administration.

3. Some rapid method of qualitative or semi-quantitative determination of fibrinogen should be used. We have used either the Schneider or the Bonsnes test.

4. The smear should be inspected for platelets.

5. A sample of the blood should be placed in a test tube and time of clot formation and degree of clot retraction should be determined. The catheterized urine is inspected for albumin. In emergency, Rh negative group 0 blood with Witebsky substance added can be administered while waiting for compatible blood. We

have gradually abandoned the use of x-rays of soft tissue or cystograms, feeling that, in general, this information is more readily and more accurately determined by physical examination. During this time no treatment has been given, no enema has been given, no internal examination has been done. The abdomen is shaved and prepared for either vaginal or abnormal delivery. Further decisions, as explained shortly, will depend on the diagnosis, the amount of bleeding, and the maturity of the fetus.

Table II which follows shows some of the clinical findings.

TABLE II

Clinical Differences Between Various Types of Bleeding

	Premature Separation Placenta	*Placenta Previa*	*Marginal Sinus*	*Vasa Previa*	*Cervical Causes*
Pain	+	0	0	0	0
Shock	+	±	0	0	0
Fetal Distress	+	+	±	+	0
Fetal Death	±	0	0	±	0
High Station of Head	0	+	0	0	0
Abnormal Presentation	0	+	0	0	0
Previous Toxemia	±	0	0	0	0
Low Fibrinogen	+	0	0	0	0
Increased Tonus	+	0	0	0	0

INDICATIONS FOR IMMEDIATE ACTION

These might be listed as: 1. Those which are present prior to the sterile vaginal examination. 2. Those which are detected on sterile vaginal examination.

Prior to the sterile vaginal examination, the persistence of bleeding, the presence of shock, the presence or appearance of pain and tender areas in the uterus, evidence of fetal distress or a tonic uterus constitute indications for immediate action which usually should be Cesarean Section. Some of the other signs which suggest the need for immediate action, are the presence of protein in a catheterized urine specimen, a marked decrease in urinary output or other evidences of toxemia.

Several contraindications to immediate action exist. The first

is severe shock. Transfusion and other corrective measures should be started immediately. Here however, we are dealing with one of the most delicate balances in medicine. Namely, when is the optimal time to perform surgery in the presence of shock, inasmuch as we know that the shock is most likely due to the continuation of bleeding. Everyone has different criteria, but in general the presence of blood transfusion running adequately with no further decrease in blood pressure and a systolic blood pressure stabilized about 80-90 mm. of mercury are sufficient indications to procede with surgery. The second contraindication to immediate action is severe anemia. Such a patient is a very poor operative risk because of the interference with the oxygen carrying mechanism of hemoglobin. Transfusion of whole blood or packed red cells should be given immediately to correct this grave deficiency and so make the patient a better candidate for anesthesia. A third contraindication is the presence of a low fibrinogen. This can be readily replaced by fibrinogen which is now available commercially. Most areas are now setting up fibrinogen banks which act as a central depot from which fibrinogen can be obtained. To operate prior to proper restoration of the normal fibrinogen level is merely to increase the bleeding and to subject the patient to grave risk. A fourth contraindication to immediate action applies primarily to placenta previa where bleeding has started while the baby is still very premature. Under these conditions expectant treatment is followed to permit the further growth and maturation of the baby; the policy at all times being to let the baby remain in utero until severe bleeding forces some method of delivery. These patients are usually best observed in a hospital in the immediate vicinity of the delivery floor so that if further bleeding occurs it can be promptly treated by transfusion and delivery. When sterile vaginal delivery is performed the findings help us reach a decision as to management. The technique of performing sterile vaginal examination should be as follows. It should be done in a delivery room which is equipped for either vaginal or abdominal delivery. It should be done with a surgical team and an anesthesiologist available. The patient should have received some Atropine as pre-medication in case anesthesia is necessary. An infusion should have been started in the arm with a large 15 or 18 gauge needle so that blood can be readily added to it. Occasionally it is desirable to have a pediatrician in attendance in case a depressed or a premature baby should be delivered. This examination may be done under anesthesia, but usually this is not necessary. The cervix may be inspected by inserting a large speculum into the vagina. At this time we may see a lesion on the cervix which apparently is causing the bleeding; or we may see a clean cervix; or we may see bleed-

ing emerging through the cervical os. Bimanual examination is then performed in an effort to determine the amount and origin of bleeding or to determine the presence of a lesion of the cervix. The length and dilatation of the cervix are carefully measured. If dilated, a finger is then inserted inside the cervical canal to determine the location of the placenta. The membranes are felt to see whether they are intact or ruptured. The presenting part is determined to be breech or vertex. If vertex, the approximate position is ascertained. At this time depending upon these findings decision is reached either to do one of five things. 1. Nothing at all. 2. Biopsy of cervix. 3. Rupture the membranes. 4. Await vaginal delivery. 5. Perform Cesarean Section. These decisions are reached upon findings. Table III suggests the various types of treatment which may be undertaken.

TABLE III

Treatment of Abnormal Bleeding at Term

Cause of Bleeding	Treatment
Minor	
1. Cervical lesion.	Biopsy, cauterization. Discharge next day.
Major	
1. Bleeding	
a. Placenta previa.	
1. Premature	No treatment
2. Mature	
a. Unripe cervix	No treatment
b. Ripe cervix	
1. Partial previa	Rupture membranes. Vaginal Delivery
2. Total previa	Cesarean Section
2. Premature separation of the placenta	Cesarean Section

Not all premature separations of the placenta need be treated by Cesarean Section. Very mild and unrecognized premature separations are usually not treated in any way, except perhaps by expectant treatment consisting of rupture of the membranes and a slow intravenous administration of oxytocin solution. We have, however, abandoned the expectant treatment of premature sepa-

ration of the placenta for anything other than the mildest of separation for the following reasons. The degree of placental separation may increase during the period of observation. The fetal heart may be lost with the resultant loss of the baby. Hypofibrinogenemia may increase; and finally at the time of delivery it is impossible to detect a uterus of Couvelaire type. For these various reasons I prefer to perform Cesarean Section whenever possible on patients with premature separation of the placenta.

OPERATION

The choice of anesthesia is very important. In general, local anesthesia consisting of abdominal wall block by 1% procaine is most preferable. This decreases further blood loss, does not increase the shock, and gives the baby a maximum chance of survival. Caudal or epidural anesthesia, while desirable from these two viewpoints, takes too long to administer to be effective. Spinal anesthesia is most undesirable because it may further increase the hypotension already caused by shock. If general anesthesia is necessary cyclopropane or ethylene seem to be most desirable because of the rapidity with which a surgical degree of anesthesia can be obtained.

In placenta previa, a low-flap type of incision is performed. This is usually the transverse incision despite the opinion of some authorities. The transverse incision is stretched by means of the fingers and the placenta if cut through, is immediately severed, the baby extracted and then as the placenta is removed it is inspected for focal areas of placenta accreta. In a rare instance a typical classical incision will be done but it is felt that the healing is so much better in a lower uterine segment that this operation is performed whenever possible. In premature separation of the placenta occasionally a classical section will be done because of the need for speed, because of shock and imminent danger to the baby; and also because the lower uterine segment has not been formed. After baby and placenta has been removed the uterus is always carefully inspected and decision is reached whether to preserve it or to remove it. Decision for hysterectomy is based upon condition of the wall of the uterus after it has been wrapped in hot towels and inspected for approximately 15 minutes. If the uterus shows no tone, if the slightest touch of the finger disrupts the wall of the uterus there is no question but that this represents a massive extravasation of blood throughout the fibers of the myometrium and such a uterus must be removed. However, only about 15-20% of uteri with premature separation of placenta need be removed. The vast majority of such separation do not result in infiltration of the wall of the uterus.

POSTPARTUM MANAGEMENT

The postpartum management of these patients, regardless of the cause of the bleeding, and regardless of vaginal or abdominal delivery, is basically the same. In the immediate postpartum period the patient is observed for signs of shock and continuation of bleeding. Pulse and blood pressure are determined. A slow infusion of intravenous oxytocin is given to keep the uterus contracted. The later management consists of inspection of the vagina and the surgical dressing for bleeding which might be indicative of the appearance or continuation of a low fibrinogen state. The patient is given iron by mouth or saccharated oxide of iron by vein to restore her hemoglobin levels. Hemoglobin, red blood cell count and hematocrit are checked on the third day to further determine the need of blood replacement.

SUMMARY

The causes of abnormal bleeding at term have been divided into minor cervical causes and major placental causes. Suggestions have been made that in case of placenta previa, more Cesarean Sections should be performed and that expectant treatment should be followed in the interest of the premature fetus. Further suggestions have been made in the case of premature separation of the placenta that the low fibrinogen state should be corrected prior to any surgery and that the tendency to increase use of Cesarean Section as opposed to vaginal delivery is a very desirable trend. Some of the outmoded procedures which I have not even mentioned, but which are now going into the discard, include: 1. The preliminary rectal or vaginal examination in the home, prior to the determination of the exact cause of bleeding. 2. Vaginal tamponade which at best is a temporary measure. 3. Manual dilatation of the cervix which greatly increases the amount of bleeding. 4. The use of Voorhees' bags or other hemostatic bags. 5. The use of Braxton-Hicks version which like all other version-extractions is accompanied by a very high maternal mortality rate. 6. The attempt to perform vaginal delivery when there is a serious degree of placenta previa. 7. Another out-moded procedure is the use of operation for delivery in the case of placenta previa regardless of the degree of maturity of the fetus. 8. The out-moded procedure of immediate Cesarean Section for premature separation of the placenta without a pre-operative evaluation of Fibrinogen.

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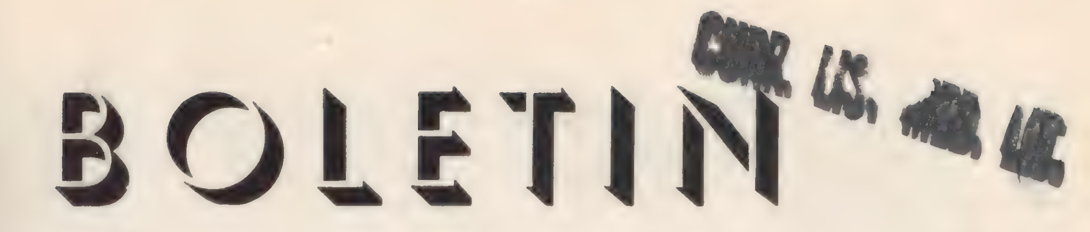
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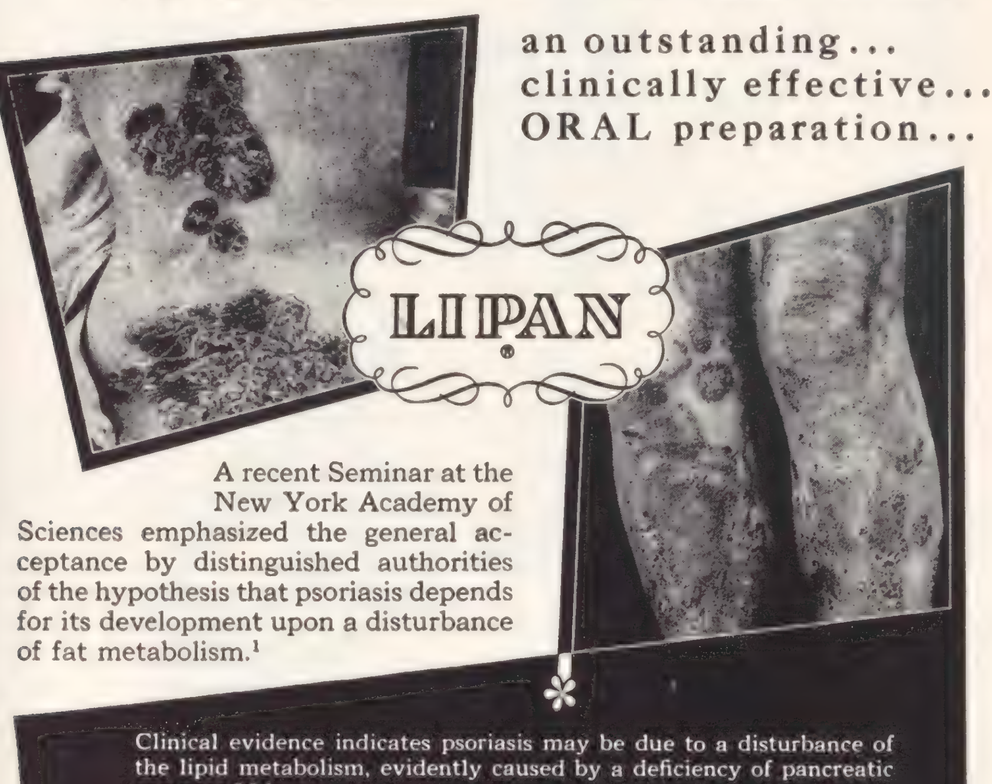
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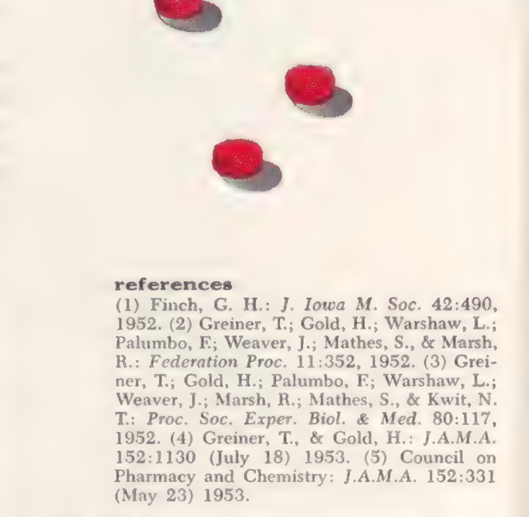
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CORTISONE IN SEXUAL PRECOCITY: A CLINICAL EXPERIMENT*

*AGUSTIN M. DE ANDINO, JR., M.D.***

and

*HECTOR O. HIDALGO, M.D.****

Normal pubertal development is caused by secretion of steroid sex hormones —testosterone from the Leydig cells of the testicles and estrogens from the follicles of the ovary. At about the same time adrenocortical 17-ketosteroid production increases; for this event, Albright has coined the term "adrenarche". It has been suggested by several students in the field that perhaps the adrenocortical 17-ketosteroids are responsible for the pubertal growth of pubic and axillary hair. There is evidence that the gonads, at least in the male, are capable of secreting androgenic hormone at an earlier age if they are stimulated by exogenous gonadotrophic hormone. This occurs incidentally in boys treated with such hormone preparations for cryptorchidism, if the dosage is too high or if treatment is unduly prolonged. This undesirable side effect, which should be avoided, indicates that the male, and probably also the female gonads are ready to respond some years before function is physiologically initiated. Probably the physiologic stimulus comes from a more or less sudden release of gonadotrophic hormone from the anterior pituitary. If the urinary gonadotrophic secretion of children approaching puberty is examined, one will find large amounts of these hormones. This phenomenon does

From the Department of Medicine and Pediatrics, School of Medicine, University of Puerto Rico.

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** Assistant Professor of Medicine, School of Medicine, University of Puerto Rico.

*** Clinical Instructor in Pediatrics, School of Medicine, University of Puerto Rico.

not last long; evidently the sex steroids, secreted because of the gonadotrophic stimulus, soon establish a balance with the anterior pituitary by their "retrograde action" upon the latter.

Sexual precocity in children may be divided into two main groups from the standpoint of etiology: 1) precocious puberty and 2) precocious pseudopuberty. In cases of precocious puberty the precocity involves both the secondary sex characteristics and the gonads; the latter not only increases in size, but proceed at an early age to produce mature sperm and ova. In these cases the advanced sexual development is the product of a premature production by the pituitary gland of trophic hormones which stimulate the gonads and the adrenals. Although a few of these cases will show an organic lesion of the brain as the etiologic factor, the majority of them fail to show any cause for the premature activation of the pituitary. Novak[1] has calculated that 90% or more of the cases of sexual precocity encountered in clinical practice are of this type. He refers to this group as "the constitutional type of sexual precocity" and suggests that the pituitary activation might be due to abnormal genetic factors.

In contradistinction, in the cases of precocius pseudopuberty the secondary sex characteristics alone appear early. In such cases the sex steroids causing early puberty-like changes are elaborated in the adrenal cortex or in testicular or ovarian tumors; the gonads themselves do not participate in the precocious development and their growth is considerably depressed. The precociously appearing sex characteristics may be isosexual or heterosexual in character.

In this paper we will present the results of an interesting physiological experiment performed in two cases of sexual precocity: the first one, a case of a male infant with congenital adrenal hyperplasia and slight virilization accompanied by a deficiency in salt hormone production by the adrenal cortex and the second one, a case of "constitutional sexual precocity" in a six years old male child which culminated in adrenal and testicular exploration.

Case No. 1

M. L. was first admitted to the hospital on November 23, 1950 at the age of 21 days with the chief complaint of persistent vomiting, weight loss and failure to suck. Physical examination revealed a very well developed, severely dehydrated male infant with a dusky, grayish color to the skin and complete muscular atonia. The presence of low serum chlorides on repeated examination suggested to the attending physician the possibility of adrenal insufficiency. On December 6, 1950 examination of the serum

by flame photometry revealed a hyponatremia of 111 mEq per liter and a hyperkalemia of 7.2 mEq per liter. The Thorn test performed on two occasions was positive with a failure of eosinophile response. On December 18, 1950 we saw the patient in consultation and it was our impression that the case represented one of adrenal insufficiency of the salt hormone type. The use of 0.5 mgm. of DOCA intramuscularly daily accompanied by extra salt was recommended. Shortly thereafter the 17-ketosteroid excretion was reported as 31 mgm. per 24 hours, the normal for an infant that age varying between 0 and 0.5 mgm. daily. The most logical diagnosis at this stage of the disease was congenital adrenal hyperplasia in a male infant complicated by adrenal insufficiency of the salt hormone type. Cortisone therapy was recommended with the purpose in mind of depressing excessive androgen production by the adrenals. Cortisone treatment was started on January 3, 1951 in doses of 5 mgm. daily intramuscularly. The dose was gradually increased over a few stormy months so that by early May 1951 he was finally being controlled with 2 mgm. of DOCA and 10 mgm. of cortisone intramuscularly plus 5 Gm. of extra salt daily. Prior to discharge from the hospital four 125 mgm. pellets of DOCA were implanted and the cortisone was changed to the oral form, the dose being increased to 7.5 mgm. twice daily.

He was followed at the Endocrine Clinic during the period of time between May and December 1951. The dose of cortisone was reduced to 5 mgm. twice daily by mouth. Adequate depression of the 17-ketosteroid excretion around 5 mgm. per 24 hours was attained. During this period of time slight evidence of androgenic stimulations was noted, as demonstrated by rapid body growth and increase in penile size. On December 28, 1951 the patient died in the hospital in an adrenal crisis precipitated by an acute upper respiratory tract infection. Autopsy examination revealed congenital adrenal hyperplasia in a male infant.

Case No. 2

The second case to be discussed is that of six years old white boy which was first seen by the senior author on October 6, 1952. The child was brought by his parents to the office because of precocious development. The birth and development of the child during the first three years of life had been uneventful. At the age of four the mother noticed that the child was growing faster than other children of the same age. At the age of five she observed that the right scrotal sac was fuller than the left one. During the year prior to examination both parents had noted a very rapid growth of the genitalia accompanied by a moderate gain

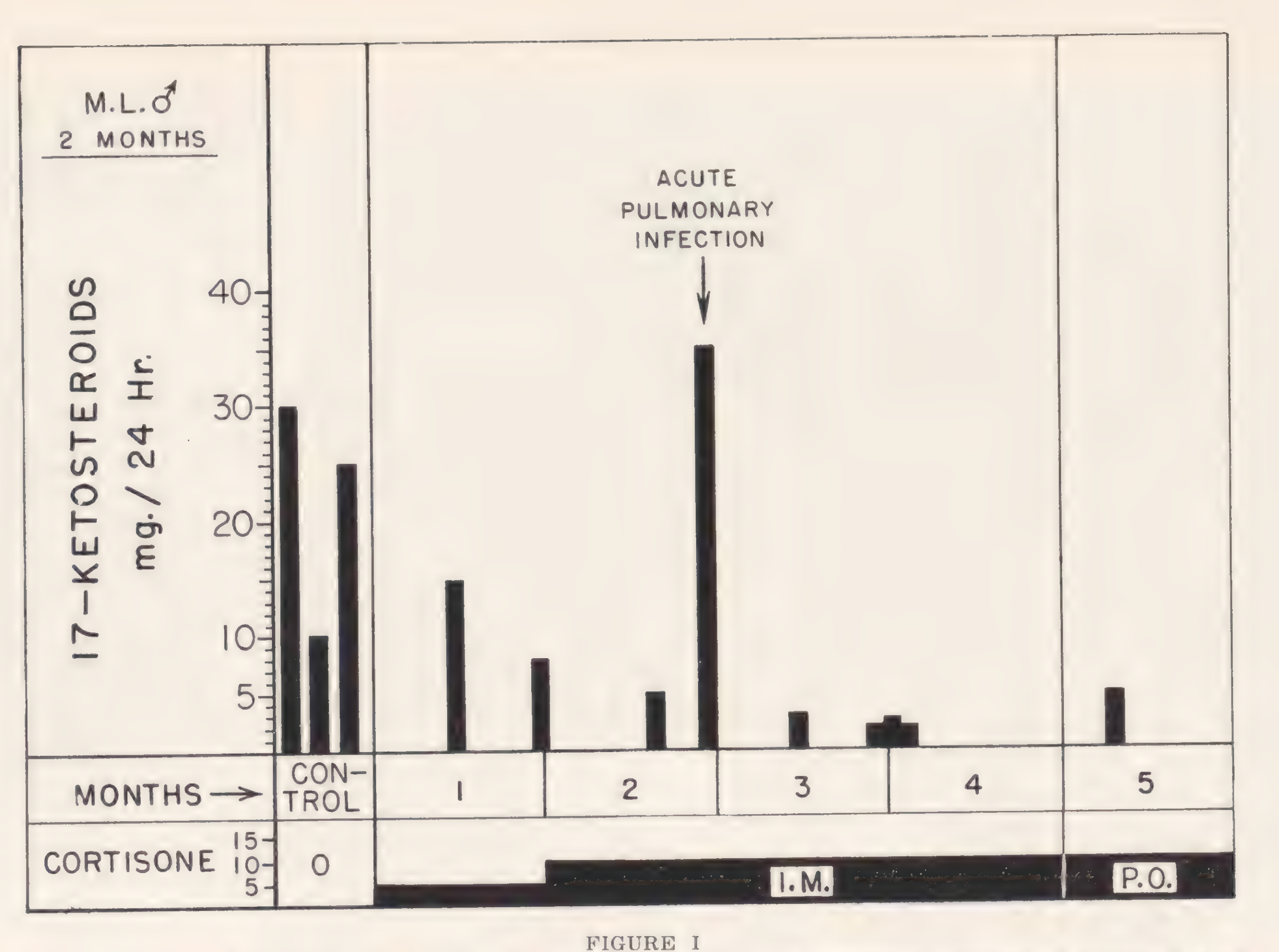


FIGURE I

This graphic chart illustrates the depressive action of cortisone upon 17-ketosteroid excretion in congenital adrenal hyperplasia (Case number One). The ability of the "cortisone-suppressed" adrenal to produce excessive amounts of steroids under stress is illustrated under the effects of an acute pulmonary infection.

in stature. Pubic hair had started to sprout three weeks prior to the first visit.

The past medical history and the family history revealed no details of significance.

The important findings in the physical examination were the following: weight, 52 lbs., height, 49¼ inches (normal for his sex and age, weight, 43 lbs. and height, 45 inches). There was slight stare to the eyes. Fundoscopic examination was normal. Examination of the abdomen was negative for organ enlargement or masses. Examination of the genitalia revealed marked development of the penis, this organ measuring 3 inches in length. The right scrotal sac was fuller than the left due to the presence of a small hydrocele. The right testicle appeared slightly indurated and irregular and was larger than the left one.

The patient was hospitalized at Rodríguez General Hospital in early November 1952. Careful laboratory studies, including X-rays of the skull, intravenous and retrograde pyelography and chemical studies of the blood were found to be within the limits of normal. The bone age was reported to be moderately advanced. The urinary neutral 17-ketosteroid excretion was reported as 9 mgm. per 24 hours, definitely elevated for a child this age, but within, normal adult levels.

On November 12, 1952 the adrenal glands were explored thru a transverse upper abdominal incision. The left adrenal gland was found to be normal in size; the right adrenal gland was found to be slightly enlarged but no tumor was palpable. The right inguinal area was explored next thru a herniorraphy incision. A congenital hernia was present which enveloped the testicle. The sac was opened and the testicle was found to be completely normal. Convalescence was uneventful.

The child was again seen on January 9, 1953 and definite evidence of continued androgenic stimulation was found. He had increased 1¾ inches in height and the penile size was 3¼ inches. Slight growth of hair was noted over the upper lip and the pubic hair had increased in amount. The 17-ketosteroid excretion on that date was reported as 14 mgm. per 24 hours.

On March 3, 1953 cortisone therapy was started in the hope of achieving depression of pituitary gonadotrophic secretion. This was given initially by the intramuscular route in doses of 25 mgm. twice daily. It was continued in diminishing doses throughout the months of March, April and May. Monthly 17-ketosteroid excretion determinations failed to reveal any evidence of pituitary-gonadal depression. Steroid administration was discontinued on May 30, 1953. The child has been seen by any one of us at regular intervals throughout this year. On the last visit there was

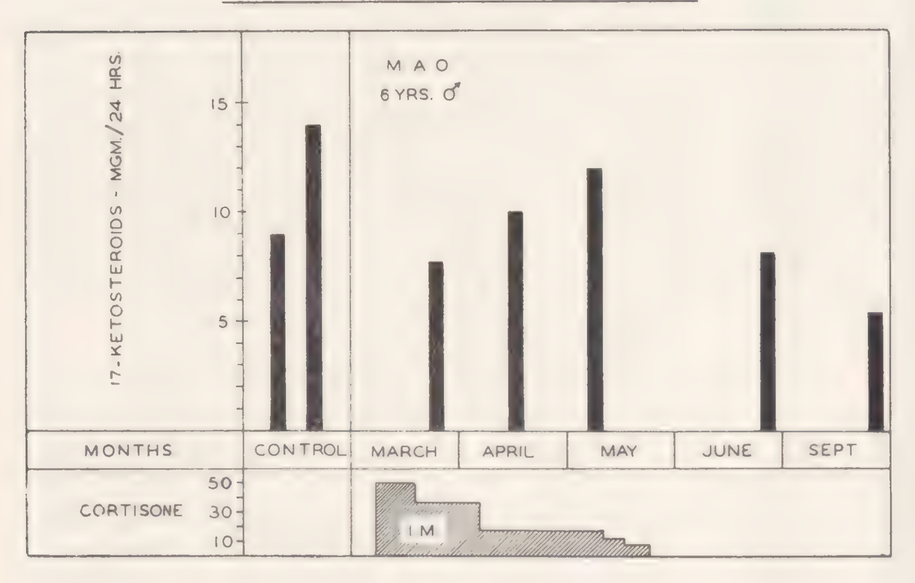


FIGURE II

This graphic chart illustrates the inability of cortisone to affect 17-ketosteroid excretion in constitutional precocious puberty of the idiopathic type (Case number Two). This is probably due to failure of depression of the gonadotrophic activity of the pituitary gland.

evidence of progressive sexual and body development. The present height is 55 inches and the weight 67 lbs. The penile length is $3\frac{1}{2}$ inches. The hair over the upper lip has increased and pubic hair is abundant.

DISCUSSION

In reference to the first case described, it is well known that congenital adrenal hyperplasia causes pseudohermaphroditism in females and macrogenitosomia precox in males. This type of hyperadrenocorticism is characterized by accelerated growth and muscular development, premature epiphyseal ossification, and early growth of sexual hair and progressive virilization, all of which are due to excessive secretion of adrenal androgen. In most of the cases described in the literature there has been no evidence of disturbance in carbohydrate or electrolyte regulation; in a few of these cases there is a disorder of electrolyte regulation causing

symptoms similar to those of Addison's disease. Our case illustrates this point quite well.

Unsuccessful attempts had been made over a number of years to suppress the secretion of androgens in patients with congenital adrenal hyperplasia by the administration of steroids, such as 17-ethyltestosterone, 17-vinyltestosterone and 17-methylandrostenediole, all of which have a chemical structure similar to that of the androgens but possessing relatively little androgenic activity. The use of cortisone in the therapy of this condition was started by Wilkins and his group[2] at the Harriet Lane Home of Johns Hopkins in June 1950 and up till the present date they have reported observations in a rather large group of patients treated continuously in periods up to about 2½ years.

Experimental studies have been carried out by various investigators to determine the mode of action of cortisone in suppressing the hyperactive adrenal cortices. Lewis and Rosemberg[3] administered 1.25 mgm. of cortisone daily for twenty days to normal rats and to hypophysectomized rats treated with ACTH. In the normal rats the cortisone caused a reduction in the size of the adrenal cortices; in the hypophysectomized rats maintained under ACTH there was no effect noted in the adrenal weight. These experiments suggest that the action of cortisone in such cases as the one described in this paper in thru the pituitary gland, but they do not imply that congenital adrenal hyperplasia is due to excessive secretion of ACTH by the adenophypophysis rather than to a primary disorder of the adrenal gland. The action of cortisone appears to be thru the suppression of output of pituitary ACTH, thereby causing marked diminution in the secretion of the pathological adrenals. This suppressive effect appears to be more relative than absolute as evidenced by the ability of the "cortisone-suppressed" adrenal to produce excessive amounts of steroids in response to the stress of an acute illness. It is quite interesting to note that the abnormal adrenal suppressed by cortisone responds more violently to the stimulus of endogenous ACTH liberated under acute stress than it does to the administration of exogenous trophic hormone.

In the type of precocious development illustrated by our second case diligent search fails to reveal any abnormality in the nervous or endocrine system. The entire pubertal mechanism is normal except for its timing. The sexual development progresses along an entirely normal pattern. Spermatogenesis occurs in boys, and in girls, true ovulatory menstrual cycles with a progestational phase may be established so that pregnancy is possible. The most famous case in medical literature, reported by Escomel,[4] is that

of Lina Medina, the Peruvian girl, who menstruated at 8 months and in 1939, at 5½ years, gave birth by Caesarean section to a 6 pound boy.

As in other types of sexual precocity, the adolescent spurt of general body growth and osseous development occurs early. During this period these patients become much taller than normal children of the same age. Because of the fact that mental and psychosexual development lag behind their physical growth, these children have to cope with real problems of social adjustment. The physician ought to be of great help in giving valuable advice and guidance during the trying years in which sexual maturation is so much far advanced than mental development.

Attempts have been made to suppress excessive activity of the pituitary gland by the administration of exogenous sex hormones or by X-ray irradiation. These procedures have met with failure and have caused more harm than good. We have reported our attempts in trying to suppress excessive pituitary gonadotrophic activity thru the use of cortisone which, as illustrated in Figure 2, have also met with failure. On the other hand, it is our impression that this procedure might serve as a valuable adjunct in the differential diagnosis of sexual precocity due to adrenal hyperplasia and that due to idiopathic activation of the pituitary-gonadal axis when this last condition occurs early in life.

SUMMARY

1. We have reported the results of cortisone administration in two cases of precocious sexual development: one secondary to congenital adrenal hyperplasia and the other secondary to idiopathic activation of the pituitary-gonadal axis.

2. In the case of congenital adrenal hyperplasia the administration of 10 to 15 mgm. of cortisone either by the intramuscular or oral route caused a drop in the urinary 17-ketosteroids from a pre-treatment level of 30 mgm. to post-treatment levels ranging from 2.5 to 5.0 mgm. daily.

3. In the case of "constitutional sexual precocity" the administration of cortisone intramuscularly for a period of three months failed to induce any change in the level of urinary 17-ketosteroid, indicating a failure of depression of gonadotrophin production by the pituitary gland.

4. The inability of cortisone to depress 17-ketosteroid excretion in cases of "constitutional sexual precocity" might serve as an useful test in the differential diagnosis of these two conditions.

5. Treatment with cortisone offers the possibility of preventing or suppressing the progressive virilization which occurs with congenital adrenal hyperplasia and will permit development of feminine characteristics in female pseudohermaphrodites.

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A CASE OF ARACHNIDISM DUE TO THE BITE OF THE BLACK WIDOW SPIDER (LATRODECTUS MACTANS) FROM VIEQUES, PUERTO RICO

*IRVING FOX, PH.D.**
*WILLIAM J. GRACE, M.D.***
*JOHN P. DEBBINK, C.E.****

The black widow spider, **Latrodectus mactans** (Fab), has been found occasionally in Puerto Rico by entomologists[1], but it leads so obscure an existence that many physicians are unaware of its existence here. Even the country-folk, who are most likely to come in contact with this spider often do not know of it, as evidenced by the fact there is no genuine common name for it in Puerto Rico, as there is in places where it is well known; our term **viuda negra,** no doubt is derived more from the movies and news notices, since it conveys no popular flavor. Yet there are common names for other venomous arthropods well known to the people, such as **alacrán** for scorpions, **ciempiés** for centipedes, **gongolí,** for millipedes, **araña peluda** for tarantulas, **guabá** for whip scorpions and **araña boba** for the giant crab spider, **Heteropoda venatoria.**[2]

Since the syndrome produced by the bite of the black widow spider may simulate appendicitis and other acute conditions (surgical operations have been performed in several cases[3]), and is of itself rather serious, particularly in the case of children, our main purpose in presenting this article is to alert the medical profession to the presence of this kind of arachnidism in Puerto Rico. In an effort to find out whether any cases of this disease had heretofore occurred, various distinguished physicians of long experience in Puerto Rico were consulted, among them Dr. Enrique Koppisch (School of Medicine), Dr. O. Costa Mandry (Department of Health), Dr. Rafael Rodríguez Molina (Veterans' Administration), Dr. Mario R. Delanoy Molina (Vieques Public Health Center), and Dr. Rafael A. Timothée (Department of Health), each of whom indicated that he had no knowledge of there ever having been a case of black widow spider bite within the political limits of Puerto Rico.

Recently, interest in arachnidism has been great and the li-

* Lt. Col., MSC (USAR), Asst. Professor of Medical Entomology, School of Medicine, School of Tropical Medicine, San Juan, Puerto Rico.

** Major, MC, Asst. Chief, Medical Service, Rodríguez Army Hospital, Fort Brooke, Puerto Rico.

*** 1st Lt., MSC, Acting Chief, Preventive Medicine Service, Rodríguez Army Hospital, Fort Brooke, Puerto Rico.

terature has become quite extensive, there being recorded no less than 50 references to articles dealing with various aspects of the disease in the Current List of Medical Literature since 1950. There seems to be some evidence for the belief that the disease has lately become more common throughout the world.[4] In the last few years useful reviews of arachnidism and its various aspects have been published by Maretic and Stanic[4], and Odom and Capel[3], and papers dealing especially with treatment have been published by Maretic[5] and Greer[6]. Gertsch[7] has published an excellent book on American spiders in general which includes interesting entomological information on the black widow.

In recent years the small island of Vieques (area 51.5 square miles), located nine miles to the east of Puerto Rico and in the political limits of the latter, has been the scene of large scale maneuvers by the U. S. Marines during the spring of each year. In April, 1956, several thousand marines participated in the maneuver, which involved an amphibious assault, simulating actual battle conditions with great reality. On April 21, 1956 the personnel at Rodríguez Army Hospital were alerted to receive a marine who was to be evacuated by emergency helicopter from Vieques with the diagnosis of arachnidism due to the bite of the black widow spider. On arrival the marine was prostrate; he had received palliative medication (demerol), and treatment with calcium gluconate had been started in Vieques. The commander of Rodríguez Army Hospital, Colonel John T. B. Strode, as well as several other medical officers, had considerable experience with arachnidism in continental United States and confirmed the diagnosis clinically. Notwithstanding the clinical evidence some doubt existed because the patient had not seen the spider, and it was known that the black widow is not very common in Puerto Rico.

It was decided that an on the spot investigation be made to find the spiders, thus removing any doubt concerning the diagnosis. Accordingly, the Army Preventive Medicine Officer (J.P.D.) and the Army entomologist temporarily on duty (I.F.) were ordered to Vieques for this purpose on April 23, 1956. Information from the patient directed the search to a place know as "Blue Beach", a supply point for off-shore Navy vessels. The patient had stated that he was bitten at about 5:00 A. M. while sleeping on an air mattress on the ground near a mortar emplacement about 100 yards inland. The area was located and companions of the patient were questioned. One remembered vividly that black widows were very common and were often found in the helmets and packs of the men in the morning. A search of the area was made and in about one-half hour five black widow spiders were

found running over the ground and under dead leaves associated with members of another genus of spiders (**Lycosa**). There were no buildings in the area or other structures offering typical places for spiders to build webs. Several rubbish piles offered suitable habitats and in one of these several specimens were found.

The spiders captured (three females and two immatures) were obviously members of the genus **Latrodectus** for they had a prominent red hour-glass like marking on the ventrum of the abdomen; but the coloration of the dorsal aspect of the abdomen was peculiar, consisting of a broad red interrupted stripe on a black background, and also the habitat was considered unusual for American black widows. Since it was important to have the species determined accurately, a female specimen was sent to Dr. Willis J. Gertsch, Curator of Arachnida in the American Museum of Natural History, New York. Dr. Gertsch agreed that the species is in fact **Latrodectus mactans**, and the specimen was deposited in the collection of the American Museum.

In 1925 Dr. Alexander Petrunkevitch visited Puerto Rico and made an extensive study of the spider fauna.[8] He found the black widow not very common and mentions that it lived in webs spun in the crevices of rocks, hollows of trees, on shrubs and cacti, in bamboo fences and in dark corners of buildings and on walls. Mr. Jorge J. Serrallés who, during the period of the Second World War, made the study of spiders his hobby, stated that the black widow was generally distributed throughout Puerto Rico, although not very common, and that he sometimes found them in the same general habitat as the Vieques specimens, that, is, running over the ground under grass.[9] How common black widows are today, and their distribution over the mainland of Puerto Rico, is not known. Aside from the site of the captures discussed in this paper, nothing is known concerning their distribution over the island of Vieques in general. To find out how abundant black widows are at the present time in Puerto Rico and their importance as a menace to the public health would require thorough field studies by trained entomologists throughout the mainland and Vieques over a long period of time, and such work could not be accomplished without more financial support than is now available. A project of this nature would undoubtedly be justified if another case should occur. Since the black widows found in Vieques were exceedingly abundant at a supply point close to the beach it is considered not improbable that they were introduced with supplies from Navy ships. These vessels originated at the Le Jeune Marine Training Area, North Carolina, where black widows are common. Since no cases have heretofore occurred, it is al-

so possible that a more vicious strain was introduced into Vieques. However, it must be admitted that Mr. Serrallés does not agree with this point of view and believes that the spiders were always present on Vieques.

Black widow spiders are very easily recognized. The mature female usually measures about one half inch in body length exclusive of the legs and is jet-black with a conspicuous red marking in the form of an hour-glass on the ventral aspect of the abdomen. Sometimes there are also red spots dorsally on the abdomen, and this was the case as regards the specimens from Vieques, one of which is shown in Figures 1 and 2 (markings shown in white in the photographs are bright red in nature). When compared with a specimen from Algiers, Louisiana, collected by E. H. Hinman[10], April 7, 1937, shown in Figures 3 and 4, it will be seen that the Vieques specimen is smaller, its hour-glass is larger and its dorsal marking are more pronounced. The Vieques specimen, although apparently a fully mature female, measured only 9 mm. in its body length and had an abdominal width of 4 mm.; the Louisiana specimen is much larger, having a body length of 12 mm. and an abdominal width of 7 mm.

Authorities differ in regard to the best method of control of black widow spiders. Gertsch[7] advocates periodical eradication of the spiders and their egg masses by mechanical means. This method might be successful when used against spiders inhabiting buildings but would be rather difficult to accomplish in places such as Vieques, and would involve an undesirable hazard for workers. Meretic and Stanic[1] believe the control of black widows by insecticides to be rather problematical and expensive and categorically state that DDT is not suitable; however, Furman and Kurtpinar[11] claim that lindane at 18 mgm. and DDT at 100 mgm. per square foot of surface provide control and protection from reinfestation for over 12 weeks and 6 weeks respectively. Keegan **et al**[12] state that DDT in spray, aerosol and powder form was effective in destroying **Latrodectus geometricus** on Luzon. In a situation such as Vieques a survey should first be made to ascertain the extent and density of infestation, and thereafter thorough applications of DDT in kerosene might be tried.

CASE REPORT

The patient is a 20-year-old marine enlisted man admitted to the hospital complaining of abdominal pain. He awoke at 5:00 A. M. feeling the sting of an insect bite over the right shoulder blade. He tried to go back to sleep but 10 to 15 minutes later



EXPLANATION OF PLATE

Figure 1. The black widow spider, **Latrodectus mactans** (Fab), female, dorsal view, from a specimen collected at "Blue Beach", Vieques Island, Puerto Rico, on April 23, 1956 by J. P. Debbink and I. Fox.

Figure 2. The same, ventral view.

Figure 3. **Latrodectus mactans** (Fab), female, dorsal view, from a specimen collected at Algiers, Louisiana, on April 7, 1937 by E. H. Hinman.

Figure 4. The same, ventral view.

began to experience severe pain in the abdomen, back and down both legs. He reported to the Naval Dispensary at Vieques, where a diagnosis of black widow spider bite was made, primarily on the basis of a rigid abdomen. The patient was immediately transferred from Vieques to Rodríguez Army Hospital. He arrived at the hospital approximately five hours after the bite, and although he had been given 20 cc. of calcium gluconate (10% solution) and 100 mgm. of demerol, he still complained of severe abdominal pain.

On physical examination at the time of admission the patient was in acute distress. The blood pressure was 140/90 He was perspiring profusely, and from time to time would have spasms of the muscles of the extremities; however, he was entirely clear headed. The actual site of the insect bite could not be found. The muscles of the abdominal wall were rigid and "board-like." The patient experienced pain on palpation of the abdomen. There was some spasticity of lumbar muscles and of the muscles of the arms and legs. The reflexes were equal and active. There was no difficulty in breathing.

Following admission to the hospital the patient was given 25 mgm. of ACTH in 1000 cc. of water. This infusion was begun at 10:00 A. M. At 1:00 in the afternoon the patient had improved remarkably and he was able to sit on the edge of the bed. His abdomen at this time was soft. That evening he was given 10 mgm. of ACTH. During the night he sweated profusely, requiring a change of clothes approximately eight times. The following day he was started on cortisone 50 mgm. every six hours. During this second hospital day he continued to perspire freely. There was no further recurrence of abdominal pain or rigidity. By the third hospital day he was completely well and the cortisone was withdrawn. He was ready to return to duty on the fourth day after the bite. He was afebrile throughout. The laboratory studies revealed no special abnormalities, and he had no eosinophilia.

DISCUSSION

The patient presented the classical clinical manifestations of black widow spider bite. The history of the insect bite, the board-like abdomen, the pain and muscle spasm in the back and legs were all quite typical. Even without the history of insect bite, the pain in the back and in the legs and stiffness of the muscles in these regions would make one feel that some process other than perforated abdominal viscus was present. However, since the black widow is not common in Puerto Rico, and arachnidism has not been reported from this Island, x-rays of the abdomen in the up-

right position were made for possible ruptured viscus. Many patients have a significant eosinophilia, and this finding is often helpful in making the diagnosis, but this sign was not present in our case. Perhaps of interest to military physicians is the fact that air evacuation by helicopter, a trip of approximately 40 minutes, seemed to have no ill effect on the patient, as attested by the medical officer accompanying the patient.

Various treatment programs have been utilized in the management of arachnidism. Probably the best known is the intravenous use of calcium gluconate in doses of 10 to 40 cc. In mild cases this medication seems to be of some benefit, although not so in our patient. Demerol in large doses is capable of alleviating the pain, and there is no reason to withhold it. An antivenom has been prepared and is said to be quite effective, but it is not generally available. Epinephrine in doses of 0.5 cc. every 3 to 4 hours has been found to be useful. This drug probably works through the pituitary-adrenal axis.

Recent reports indicate the prompt relief of symptoms following the administration of cortisone and ACTH. Sucessful treatment has resulted from the use of as little as 80 mgms. of cortisone[5], but a much higher dose would be necessary for a severe bite. ACTH is usually used in doses of 10 to 25 mgms. in 1000 cc. of 5% glucose in water, once or twice daily.[13]

SUMMARY

The black widow spider **Latrodectus mactans** (Fab) occurs in Puerto Rico, but no case of arachnidism due to its bite has heretofore been reported. During the 1956 maneuvers of the U. S. Marines on Vieques Island, P. R., a case occurred. An inspection of the place where the marine acquired the disease showed that black widows were very common on the ground under dead grass, but whether they had been introduced with supplies from continental United States or were already present is not known. On admission to the hospital the patient had a "board like" abdomen and spasticity of the muscles of the back and extremities. He sweated profusely at night. Demerol alleviated his pain. Calcium gluconate was not very beneficial. Treatment with ACTH and then cortisone resulted in prompt relief of symptoms and full recovery by the fourth day after the bite.

RESUMEN

La araña "viuda negra", **Latrodectus mactans**, existe en Puerto Rico, pero ningún caso de aracnoidismo había sido informado hasta ahora. Durante las maniobras de la Infantería de Marina de

los Estados Unidos en la isla de Vieques, P. R., en 1956, se observó un caso. Una inspección del sitio donde el marino sufrió la picadura demostró que las viudas negras eran comunes en el suelo debajo de la yerba seca. Se desconoce si éstas fueron introducidas con los abastecimientos traídos de los Estados Unidos o si ya existían en Vieques. Al ingresar en el hospital el paciente mostraba rigidez abdominal y espasticidad de los músculos de la espalda y extremidades. Perspiró profusamente durante la noche. El dolor se alivió con demerol, pero el gluconato de calcio no fué muy efectivo. El ACTH y la cortisona le aliviaron y el paciente fué dado de alta cuatro días después de la picadura de la araña.

ACKNOWLEDGMENTS

Thanks are expressed to Dr. Enrique Koppisch, School of Medicine, School of Tropical Medicine, University of Puerto Rico, San Juan, P. R., for his helpful advice and reading the manuscript. Thanks are also due Col. John T. B. Strode, M. C., Commanding Officer, Rodríguez Army Hospital, Fort Brooke, P. R., for his continuous encouragement and facilitating the expedition to Vieques Island.

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PRACTICAL USE OF X RAY PROCEDURES IN OBSTETRICS

EDWARD O'NEILL, M.D.
Santurce, P. R.

Roentgenography is a valuable aid to the obstetrician. Although it has been made use of for a long time, it has not been until recently that textbooks of obstetrics have given it a chapter of its own. At first the X-ray apparatus were inadequate for use in obstetrics. However, with time, the apparatus have been modified and improved and therefore have become more reliable.

Roentgenography is a help in the diagnosis of pregnancy. However, its use is limited by the fact that until the 4th month of life the fetus cannot be visualized. It is true that by the time the fetus is visualized by X-ray it can also be heard. However, in cases in which there is doubt between pregnancy and a fibromyoma of the uterus, a negative X-ray for presence of a fetus plus a negative pregnancy test provide adequate protection not only for the patient but the physician also. In cases in which a hydatidiform mole is suspected the presence of a fetus will eliminate that possibility.

It is well to remember that the inability to demonstrate a fetus is not absolute proof that a pregnancy doesn't exist. The answer may be that the pregnancy is of less duration than expected, or that obesity of the abdominal wall or a fibromyoma of the uterus interfere with visualization of the fetus.

In certain obese patients it is not easy to determine the presentation or position of the fetus; or whether it is a single or a multiple pregnancy. A flat plate of the abdomen will help in these cases. Occasionally in cases of suspected multiple pregnancy, a lateral X-ray is also necessary for one fetus may hide behind the other.

The diagnosis of bony fetal abnormalities can be established by X-ray. It is indicated in those patients in whom the possibility of a fetal abnormality exists, namely: diabetic mothers, patients with polyhydramnios, patients who have given birth to an abnormal baby previously, sensitized Rh negative mothers, the very young or the very old primigravida. Hydrocephalus can be diagnosed not only by the size of the head, but also by the small size of the face as compared to the skull, by the thinness of the bones, and by the size of the fontanelles. It is well to look at the baby for evidence of spina bifida, for one third of the cases of spina bifida are associated with hydrocephalus.

A halo around the fetus means edema of the soft tissues. This can be seen in cases of hydrops fetalis. Also in babies of

diabetic mothers. It is of the same density of amniotic fluid, however, it causes obliteration of the subcutaneous fat. Heavy accumulation of subcutaneous fat occur in babies of diabetic mothers.

Diagnosis of fetal death can be confirmed by X-ray. Spaulding sign (marked overlapping of cranial bones) is the most common sign seen. This is due to the loss of fluid and collapse of cranial bones. It is detected as early as 3 to 4 days after death. Another sign of fetal death is sharp angulation of the spine due to loss of muscle tone.

Prematurity being the most common cause of neonatal death, its prevention should be one of the main objectives of the obstetrician. Now here can this be prevented better than in cases of repeat Cesarean section. Waiting for the patient to go into labor might be dangerous. Elective Cesarean section at a proper time is safer for mother and baby. Demonstration of distal femoral epiphysis by X-ray, means that the baby is over 36 weeks. For practical purposes, babies of 36 weeks or more do as well as babies of 40 weeks.

The most common use of X-ray is in the measuring of the pelvis.

X-ray pelvimetry is indicated in any of the following conditions:

1—All gravidas with a malpresentation.

2—Primigravida at term with floating head.

3—Patients with pelvis found small by clinical examination.

a- Promontory of sacrum easily reached.

b- Prominent spines.

c- Small transverse of outlet.

4—Patients with a history of a difficult delivery.

5—Patients with history of unexplained stillbirth.

6—Cases of dystocia, with doubt as to adequacy of pelvis.

The optimum time for this pelvimetry is when the patient is in labor, for this is when all 5 factors which determine vaginal delivery can be properly evaluated, namely size of pelvis, size of baby, uterine contractions, presentation and position and moldeability of the head. Although this is ideal, it is not practical. Patient may go into labor at hours of the night when time may be wasted getting the X-ray technician, etc.

So, in practice, pelvimetry is performed around the 38th week of gestation. There are several methods for X-ray pelvimetry: Thoms, Torpin, Colcher-Sussman, Ball. The Colcher-Sussman is very simple and easy to read by the obstetrician. We can use Mengert's quadrants as a guide as to the adequacy of the pelvis. Dr. Mengert multiplies the transverse of the inlet by the A. P. of the inlet. He does the same with the midplane. He uses as normal

145 sq. cm. for the inlet and 125 sq. cm. for the midplane. He states that disproportion exists when you have less than 85% of normal pelvic capacity. Normal values for A. P. and transverse diameters of inlet and midplane are: Inlet AP 11. 5cm trans. 12.5; midplane AP 11.5, Trans 10.5.

In the diagnosis of placenta previa, particularly in cases in which a vaginal examination is to be deferred, a placentogram or a cystogram will be of help in establishing the presence of placenta praevia. The demonstration of placenta implanted in its normal position on a placentogram is positive evidence of absence of placenta praevia. The demonstration in a cystogram of something in between bladder and the fetal head is suggestive of presence of placenta praevia. This is of value only when the placenta is in the anterior wall.

Finally X-ray is of invaluable help in diagnosing conditions complicating pregnancy; demonstration of calcifications in diagnosing a dermoid cyst of the ovary; I. V. pyelograms in establishing the status of the kidneys, ureters and bladder; Chest X-ray in helping to detect unsuspected tuberculosis. A chest X-ray or at least a microfilm should be a routine on all pregnant patients. It was shown by a study at Chicago Lying In that they had an incidence of unsuspected tbc of 1%. Our standard of living being lower than that of the people in the United States, we must have a higher incidence of unsuspected tbc. However, in practice we order it only on cases of contact, or on cases on whom the disease is suspected.

Although roentgenography is of invaluable help to the obstetrician, it is not to take the place of clinical diagnosis and clinical judgement.

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REVIEW OF MAJOR GYNECOLOGICAL SURGERY*

*DAVID HOLMES CHAFEY, M.D.,***
Santurce, P. R.

Exactly four years ago this month, the Dept. of Obstetrics and Gynecology of the San Juan City Hospital was fully approved for the full three year residency training program in this specialty. What is intended at this time is a discussion or a general review of the major gynecological surgery performed since that time and the trends that seem to be developing in our consideration of the different operations that may be performed and our reasoning for the choices made. It is not the purpose of this review to delve into the analysis of so many cases of hysterectomy, for instance, as regards age, symptomatology, indications, morbidity rates, etc.

To begin with, the specialty of gynecology appears to be one that was an advanced specialty from its very beginning. The operations of gynecology were manifold and every woman presenting the slightest evidence of pelvic illness was immediately subjected to some form of operation or other. You will note, curiously enough, that of all the specialties, gynecology has advanced least in technique. Refinements have taken place, it is true, but no new major procedures other than the evisceration operations for malignancy now being performed and evaluated, have taken place in last fifty years. Vaginal hysterectomy seems in the minds of some to be a comparatively new procedure but in reality it is only its popularity that is new. Anyone who saw Pryor, of New York, do his vaginal hysterectomy many many years ago will realize that the operation was well established almost all the instruments used in gynecological surgery bear the names of operators long since gone from this earth. The Hegar and Goodell dilators; Sims speculum; Kelly clamps; Pean hemostats, are ancient instruments, from the standpoint of modern medicine. So I repeat, it is a curious fact that operative gynecology, contrasted to some of our other operative specialties, has not advanced greatly because it reached its acme so far as is foreseeable many years ago. And in addition to this it appears as though instead of devising and practicing new operative procedures we are discarding many procedures which were at one time very popular and respected ones. It is this last point which I think will be born out to a large extent in the recent operative procedures done on our service.

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* Presented at monthly Staff Meeting, San Juan City Hospital, July, 1956.
** Assistant Professor, Dept. of Ob-Gyn., School of Medicine, Univ. of P. R.

Since removal of the uterus has become the most common major gynecological operation, it is with this topic of hysterectomy that we will dwell on the most. If you will look on the chart at columns one and two, you will see the number of abdominal and vaginal hysterectomies performed on the Obstetrical and Gynecological Service over the past four years. It is immediately obvious that the incidence of vaginal over abdominal hysterectomies has been definitely increasing so that the percentage of vaginal hysterectomies over the last six-month period was 58%. Comparing this to one of the more recent reports on hysterectomies in the literature we note that Kimbrough reports from the Pennsylvania Hospital a vaginal hysterectomy percentage of only 25%. What now might be some of the reasons for this discrepancy? Certainly the higher incidence of excessive multiparity here in Puerto Rico with its damaging effects to the supporting structures of the uterus leading to prolapse is a very important factor. Somewhat less important factors are perhaps not an overall as high a level of obstetrical care exists here as among the general population of the city of Philadelphia. Nutritional factors also probably play a part. And last but certainly not least, the broader indications for vaginal hysterectomies that seem to prevail among the attending physicians on our service. In other words, if a hysterectomy is deemed necessary on a patient, the vaginal route is first considered and a contraindication sought rather than an indication for the vaginal hysterectomy. The vagina is the natural pathway to the pelvic organs and in addition to its well established social and obstetrical functions, we seem to feel that its surgical functions should be more often utilized. For all practical purposes all the 214 vaginal hysterectomies listed were accompanied by anterior and posterior colporrhaphy and perineorrhaphy and in many instances of elderly women with procidentia, a colpocleisis was carried out. This brings us to the now important point of what might be considered the procedure of choice for a patient with symptomatic vaginal relaxations with or without uterine prolapse who has completed her family. By looking at vertical columns 4 and 5 you can readily appreciate the thoughts of our department in this respect. The incidence of simply doing a vaginal plastic or a Manchester operation is very low in comparison with the incidence of vaginal hysterectomy. And actually, of the thirteen Manchesters or Fothergill operations done, a considerable number of these were performed for teaching purposes only. Most of the others submitted to a Manchester, considering our present trend of thought, could probably have been better served by a vaginal hysterectomy with anterior and posterior repair, and the rest, along with many of the anterior and posterior repairs who were desirous of having

REVIEW OF MAJOR GYNECOLOGICAL SURGERY — DEPT. OF OBS-GYN. — JULY, 1952 - JUNE, 1956

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
	Abd. Hyst.	Vag. Hyst.	Lap.	A - P Repair	Man-chester	Subtot. Hyst.	Vul-vect	LeFort	Watk	Werth	Abd. Susp of Vag.	First Rep.	Vag. Myo.	Trach	Abd. Myo.	Bass	Recons. of Vag.	Vag. Rep. fol. Pro.
July-Dec. 1952	19	15	25	6	1	2	2	4	1	3	0	1	0	1	0	0	1	0
Jan.-Dec. 1953	56	46	32	16	2	6	0	1	1	2	0	3	0	3	0	1	1	1
Jan.-Dec. 1954	47	50	34	6	7	3	4	2	0	3	1	3	1	0	0	1	0	1
Jan.-Dec. 1955	51	63	39	14	3	3	2	2	0	2	3	2	2	4	0	0	0	2
Jan.-June 1956	28	40	23	5	0	1	4	2	0	0	0	0	1	1	0	1	0	1
Total	201	214	153	47	13	15	12	11	2	10	4	9	4	9	0	3	2	5

more children, could perhaps been given symptomatic relief with a well fitting pessary until they had completed their family and then a more complete type of operation performed.

We certainly do not mean to say that in 100% of the cases of plastic vaginal repairs done on women who desire no more children or who are past the childbearing age, a vaginal hysterectomy should be performed. There certainly will be an occasional case where the uterus is high and well supported and its removal would result in surgical gymnastics in addition to increasing the dangers of injury to the bladder or ureters, as well as the increased operating time and increased blood loss.

It has often been stated by more than one experienced gynecological surgeon that in women at the end of their childbearing period, any operation on the uterus, except diagnostic curettage, should be a hysterectomy. This surgical review seems to go along with this line of reasoning.

What all this seems to mean then is that we are narrowing down our list of major gynecological procedures by discarding some of the manifold operations that our specialty originally started out with. To name a few of these we can mention the Watkins Interposition Operation, Manchester or Fothergill or Donald Operation, Sturndorf tracheloplasty, the Spalding Richardson Composite Operation for uterine prolapse and allied conditions; the various uterine suspension operations such as the Baldy-Webster, modified Gilliam or Simpsom Montgomery or the simple ventral suspension of the uterus to the anterior abdominal wall; the Goodell-Power Modification of the Le Fort operation. You will note that uterine suspensions and myomectomies are conspicuous by their absence in this four-year review. And as regards operations for stress incontinence the various sling operations, including the Millon-Reed, Goebell-Frangenheim-Stoeckel or its Aldridge modification, are becoming less and less popular and have been completely abandoned in many clinics. We feel that the first attempt to cure stress incontinence should be a simple anterior colporrhaphy and if there is a recurrence, the Marshall-Marchetti Krants retropubic periurethral suspension operation should be utilized. We have performed this latter procedure 4 times, all in conjunction with abdominal hysterectomies. As regards the increasing number of hysterectomies, vaginal or abdominal, that our service is performing, I would at this point, like to quote Dr. Edward Schuman, who needs no introduction to physicians in our specialty. Commenting on hysterectomy he states "I object very strongly to the statement that the pathology and physical findings are sufficient criteria on which to judge whether the removal of a uterus was justified. This overlooks the third factor, how is the woman? Is she im-

proved? Has the operation cleared up her leukorrhea, her dyspareunia, the heaviness in the pelvis, the chronic recurring lower abdominal and sacral discomfort, and so on?" "The uterus is sometimes regarded as a sort of a sacred organ. Nobody questions the removal of a gallbladder, a thyroid gland or tonsils, but touch the uterus and you are committing some sort of sin."

In commenting on some other points with regards to hysterectomy and the work up of a patient prior to surgery on our service, we almost always precede the hysterectomy, vaginal or abdominal, with a preliminary D & C. And it goes without saying that there is no place for the subtotal type of hysterectomy except in a very few exceptional cases. A biopsy report of the cervix is now always obtained, prior to hysterectomy. A vaginal smear also is suitable in ruling out malignant changes in the cervix. All patients for major surgery over the age of 45, in addition to the routine blood count, serology, urinalysis and blood chemistries, are also given the benefit of a chest X-ray and an E.K.G. when possible. Any abnormal findings necessitates medical consultation prior to surgery. 500 cc of whole blood is always available in the O. R. at the commencement of the operation. Spinal anesthesia is used almost routinely. 10 mg. of pontocaine and 100 mg. of novocaine. This is often supplemented with sodium penthotal intravenously. And needless to say, all tissue specimens removed are sent to the pathological laboratory. The question of prophylactic removal of the ovaries at the time of hysterectomy for benign disease after the age of 40 or 45, is as controversial in our department as it is in any other gynecological department. In general, if the patient is over 45 years of age we remove the adnexa bilaterally.

Proceeding now to vertical column 3 we note 153 laparotomies, performed over the last four years. For the most part these were done for ectopic pregnancies, ovarian cysts and chronic P.I.D. As regards ectopic, I would like to spend just a moment to digress on how we manage our cases of suspected or possible ectopic gestation. Certainly with the more clear cut cases, if there is such a thing, with intraabdominal hemorrhage, etc, there is unanimous agreement on immediate transfusions and laparotomy. But with the suspected case there are many differences of opinion as regards the management. Some clinics utilize the pregnancy test, the culdoscope, a needle aspiration of the posterior cul-de-sac, or just follow the patient with serial blood counts every three to four hours. None of these are infallible and when you are dealing with a condition such as ectopic gestation that still has a 1.5% mortality rate, we feel you have to be 100% sure. Certainly if the pregnancy test is positive, the laboratory animal cannot tell us whether

the patient is pregnant. Anyone who has performed culdoscopic procedures can testify to the often dubious results obtained and the differences of opinion that exist among the clinicians trying to arrive at a diagnosis by looking through a narrow series of lens and lights. The cul-de-sac puncture is not without danger and if the ectopic gestation is well walled off or the blood clotted at the puncture site, there will be none withdrawn. Occasionally a blood vessel will be entered which will give a false positive test. For these reasons we prefer to do a simple posterior colpotomy using sodium pentothal anesthesia. Here the tubes can usually be easily palpated and visualized. If free blood is found or an ectopic without free blood in the peritoneal cavity, a laparotomy is immediately performed. If nothing is found, a major operation has been avoided, a dangerous condition ruled out and the patient may be discharged the following day.

In moving on to column six on our chart you will notice that only 15 subtotal hysterectomies were performed in four years. An incidence of about 6%. I dare say that all of these cervices were left behind either because the patient went bad on the operating table and it was deemed advisable to close the abdomen as soon as possible or that the cervix was so buried in adhesions that the danger of injury to the bladder and ureters plus further blood loss and operating time was greater than the danger of allowing the cervix to remain. With the incidence of stump carcinoma about 5% plus other diseases of the cervical stump that might be discomforting to the patient, it is obvious why the subtotal procedure should be avoided if at all possible.

Columns 7 and 8, listing the number of vulvectomies and Le Fort operations, do not require much comment. The simple vulvectomy is performed for leukoplakia and the radical vulvectomy for carcinoma of the vulva. In the case of the latter we feel that superficial and deep inguinal and femoral lymph node dissection should be carried out. This has been done 3 times in the last 4 years as is showed in column 16. The Le Fort partial colpocleisis operation we still feel has a very definite place in the management of complete prolapse of the uterus in elderly women who perhaps cannot withstand a vaginal hysterectomy and vaginal repair under general or spinal anesthesia. The decision as to what would be the procedure of choice or whether to be satisfied with just inserting a doughnut pessary, is usually left to the medical consultant.

I would like to close with a few words about carcinoma of the cervix. Column 10 shows that ten radical hysterectomies with pelvic lymphadenectomies have been performed in the past four years. This reflects the fact that there is as much discussion and controversy in our department as in any other gynecological de-

partment as to whether the stage I and early stage II carcinomas of the cervix cases can best be handled by surgery or irradiation. In general the majority of the staff feel that with the higher doses of radiation and improved radiotherapeutic techniques, our radiology contemporaries will give the patient a better absolute 5 yr. salvage rate than the surgical enthusiasts. The one thing we all do agree on is that our overall salvage rates are poor with either treatment and until the cause of cancer has been determined we should perhaps divert more of any energy toward possible preventative and prophylactic measures rather than arguing which of two poor types of treatment is best.

It has been said by wise men that surgery is inevitably a confession of defeat unless it is practiced in the relief of trauma. In gynecology we are still entirely too surgically minded. We cut out disease rather than attempt to prevent it. The tremendous amount of study devoted to the cause of cancer, cause of myomas, further work of antibiotics to help completely eliminate pelvic inflammatory disease, and even studies as to why the uterine and vaginal supports weaken in some patients and not in others, now going on, will inevitably bear fruit one day, and when some of these causes have been discovered, prevention and prophylaxis will rapidly become automatic, thus relieving gynecology of much of its surgery. In any event until that day arrives, the type of surgery we are performing now we feel is best serving the indigent females of the San Juan metropolitan area.

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INFORME DEL PRESIDENTE

— AÑO 1956 —

Apreciados compañeros:

Tras un año en extremo accidentado y decididamente lleno de interés, me permito presentar a esta Honorable Cámara un resumen de nuestras actividades. Gran parte de lo que he de decir es ya de conocimiento de la mayoría de los que me escuchan, pero considero beneficioso hacer hincapié en algunos de estos puntos.

Comité de Querellas

Ha sido magnífica la labor de este comité. Ha intervenido sabiamente en un número de querellas según oiremos oportunamente, y sus decisiones justas deben recibir la aprobación de toda nuestra matrícula ya que han demostrado a la comunidad que esta Asociación tiene verdadero interés en que nuestro pueblo sea bien asistido y a través de honorarios razonables.

Comité de Edificio

Tras numerosas entrevistas con el Secretario de Obras Públicas, el Secretario de Salud y la Junta de Planificación se logró interesar a estas autoridades gubernamentales en adquirir este edificio a cambio de un solar más adecuado, localizado en un ambiente más apropiado a nuestros fines, que sería vendido a nosotros a un precio módico y con título libre de restricciones. Creímos haber conseguido un espléndido solar de 3 cuerdas en la Avenida Franklin D. Roosevelt, pero estando ya en vísperas de ultimar los detalles de la transacción, la Junta de Planes interpuso obstáculos, ya que esta Junta había asignado otro uso a dicho solar. Nos vemos pues precisados a volver atrás y escoger otro solar que esté disponible. Debemos mientras tanto continuar nuestros esfuerzos de recaudar fondos para nuestra nueva casa.

Escudo Azul

Hábilmente aconsejados por dirigentes de la Asociación de Escudo Azul Nacional hemos puesto todos nuestros esfuerzos en conseguir nueva legislación que haga posible la creación de un verdadero Escudo Azul, patrocinado por los médicos, guiado por los médicos y servido por los médicos. Hemos cambiado impresiones con altos personajes de las ramas ejecutivas y legislativas de nues-

tro gobierno y todos han mostrado vivo interés en nuestros conceptos. Se nos han brindado los servicios del Comité Legislativo de la Legislatura de Puerto Rico bajo cuya dirección se está redactando el anteproyecto que habrá de deslindar los campos entre Cruz Azul y Escudo Azul. Son nuestros propósitos presentar esta nueva legislación en la próxima sesión legislativa.

Nuestra labor en esa ocasión será árdua: habrá que acercarse y convencer a los legisladores y al Ejecutivo. Vamos a necesitar de la cooperación de todos Uds., de todos nuestros médicos, para presentar nuestros puntos de vista de manera clara y convincente. Aunque el Superintendente de Seguros nos ha negado su ayuda, confiamos en obtener nuestros propósitos, ya que creemos que nuestra posición es lógica, justa y razonable.

Médicos Extranjeros

En vista de que la Ley 383, que autoriza a médicos a ejercer en Puerto Rico mediante licencia especial vence en Junio de 1957, creímos imprescindible el que se legislase para corregir esta situación anómala. Nos acercamos al Hon. Secretario de Salud para pedirle que presentase él, como proyecto de su Departamento, la legislación pertinente. Resumimos en la siguiente carta la posición de nuestra Asociación:

17 de octubre de 1956

Dr. Juan A. Pons
Secretario de Salud
Departamento de Salud
Santurce, P. R.

Mi muy apreciado Dr. Pons:

Considero muy fructífera nuestra conversasión de ayer y me permito resumir la posición de la Asociación Médica de Puerto Rico, por voz de su Junta de Directores, con respecto a la ley 383.

Es nuestra opinión que todo médico actualmente ejerciendo en Puerto Rico al amparo de la ley 383 debe someterse a exámenes de reválida antes de que se le autorice a ejercer librémente la profesión. Ya que la ley 383 expira en junio de 1957, se debe legislar fijando un plazo de seis meses a partir de esa fecha, para que dentro de ese tiempo se presenten a examen de reválida todos los que ejerzan bajo esa ley. Aquellos que sean ciudadanos americanos recibirán su licencia permanente al aprobar el examen. Se les permitirá también a

los que no hayan jurado la ciudadanía americana tomar la reválida; pero aunque aprueben ésta no recibirán, bajo ningún concepto, su licencia permanente hasta que cumplan con los requisitos de ciudadanía.

Es nuestro criterio que siguiendo estas normas conservaremos la uniformidad en el procedimiento empleado para otorgar licencias a nuestros médicos. Creemos también que el plan propuesto le facilitará el procedimiento al médico extranjero, ya que se le permitirá presentarse a reválida sin obligarlo a renunciar la ciudadanía de su país de origen, por lo que fácilmente podría regresar a él de no aprobar su reválida en Puerto Rico.

Agradezco el privilegio que Ud. me brindara de discutir estos asuntos con Ud. y espero tener la oportunidad de revisar el anteproyecto que surja de estas conversaciones.

Cordialmente,

Jaime F. Pou, M.D.
Presidente

El Secretario de Salud ha redactado el siguiente anteproyecto:

PROYECTO DE LEY

PARA ESTABLECER LOS REQUISITOS MEDIANTE LOS CUALES LOS MEDICOS CONTRATADOS EN VIRTUD DE LAS DISPOSICIONES DE LA LEY ESPECIAL PARA LA CONTRATACION DE MEDICOS, LEY NUM. 383 APROBADA EN 22 DE ABRIL DE 1946, PODRAN CONTINUAR EJERCIENDO LA PROFESION MEDICA EN PUERTO RICO.

EXPOSICION DE MOTIVOS

Las necesidades por médicos de las fuerzas armadas durante los años 1941 a 1946 redujeron el número de estos profesionales disponibles a la comunidad. Por otra parte, la expansión en los servicios médicos públicos aumentó las necesidades de la comunidad por estos profesionales.

Las leyes Núm. 26 aprobada en 10 de abril de 1942 y Núm. 29 aprobada en 29 de abril de 1943 autorizaron el ejercicio de la profesión médica en Puerto Rico a ciertos médicos que no reunían los requisitos de la ley que rige el ejercicio de la profesión médica, en algunos de los casos siendo tal requisito el de la ciudadanía.

La Ley Núm. 383 aprobada en 22 de abril de 1946 autorizó al Secretario de Salud a contratar para ciertos servicios médicos públicos los servicios profesionales de médicos que no reunían los requisitos de la ley que rige el ejercicio de la profesión médica, sujeto a ciertas condiciones; dichos médicos habrían de acreditar sus conocimientos médicos ante el Tribunal Examinador de Médicos, "incluso mediante examen" cuando así lo acordare el Tribunal. Al derogar las leyes especiales anteriores (de 1942 y 1943), convalidó las licencias otorgadas a virtud de ellas "hasta un año después de ser oficialmente declarado terminado el conflicto bélico por el Presidente de los Estados Unidos". Estas licencias han quedado, de hecho, en vigor hasta el presente.

La Ley Núm. 383 de 1946 había de estar en vigor hasta 30 de junio de 1950; enmiendas subsiguientes han extendido la duración de su vigencia. La Ley Núm 51 aprobada en 2 de junio de 1955 prorroga su vigencia hasta 30 de junio de 1957. Otras enmiendas autorizaron a los alcaldes, al Fondo del Seguro del Estado y a la Autoridad de Tierras a contratar directamente sin intervención del Secretario de Salud.

Algunos de los médicos cobijados por las leyes de 1942 y 1943 han adquirido la ciudadanía de Estados Unidos y han obtenido licencias permanentes para el ejercicio de la medicina de acuerdo con la ley que rige el ejercicio de la profesión médica, por decisiones de las cortes; a virtud de esas mismas decisiones las obtuvieron también otros que siempre fueron ciudadanos de Estados Unidos. A virtud de la Ley Núm. 383 de 1946 fueron autorizados otros sin examen o con breves exámenes orales; más recientemente, algunos han sido sometidos, a los fines de la autorización provisional, al examen regular que el Tribunal examinador de médicos ofrece a los aspirantes a licencia permanente de acuerdo con la ley que rige el ejercicio de la profesión médica. Muchos de los médicos originalmente autorizados a ejercer provisionalmente a virtud de las leyes especiales han adquirido luego la ciudadanía de Estados Unidos, se han sometido a examen regular, lo han aprobado y ostentan ahora licencias permanentes.

Esta ley tiene el propósito de ofrecer amplias oportunidades a los médicos que todavía ejercen a virtud de las leyes especiales, de afianzar su posición como profesionales ante la comunidad para que puedan eventualmente ejercer su profesión libremente, sosteniendo el principio de ciudadanía, que establece la ley que rige el ejercicio de la profesión médica y colocándolos, a los fines de acreditación de sus conocimientos, en condiciones iguales a las que privan con relación

a los que normalmente se gobiernan por la ley que rige el ejercicio de la profesión médica.

DECRETESE POR LA ASAMBLEA LEGISLATIVA DE PUERTO RICO:

ARTICULO 1.—**Definiciones.** Los siguientes términos se entenderán en esta ley en el sentido que aquí se establece:

A. **Ley del ejercicio de la profesión médica:** La Ley Núm. 22 aprobada en 22 de abril de 1931, "Ley para regular el ejercicio de la profesión médica en Puerto Rico; para establecer el tribunal examinador de médicos;..." etc. según ha sido subsiguientemente emendada. Incluye también sus reglamentos.

B. **Ley especial para la contratación de médicos:** La Ley Núm. 383 aprobada en 22 de abril de 1946, "Ley para autorizar al Comisionado de Sanidad a contratar los servicios profesionales de médicos ciudadanos americanos o extranjeros, a fijar las condiciones de dichos contratos de servicios;..." etc. según ha sido enmendada.

C. **Convocatoria ordinaria:** La que hace el Tribunal Examinador de Médicos para celebrar exámenes regulares, dos veces al año, de acuerdo con el artículo 10 de la ley del ejercicio de la profesión médica (definición A) y que se celebran en marzo y setiembre de cada año.

D. **Exámenes regulares:** Los que celebra el Tribunal Examinador de Médicos dos veces al año de acuerdo con la ley del ejercicio de la profesión médica (definición A) por convocatoria ordinaria y de acuerdo con sus reglamentos para esa Ley.

E. **Tribunal:** El Tribunal Examinador de Médicos establecido por la ley para el ejercicio de la profesión médica.

F. **Licencia Permanente:** La que otorga el Tribunal a virtud de la ley del ejercicio de la profesión médica; cuando se dispone el otorgarla a virtud de esta ley, se entiende que queda sujeta a todas las condiciones sobre cancelación, etc. que aquella ley establece.

G. **Licencia provisional especial:** La licencia o autorización que el Tribunal otorga a virtud de la Ley especial para la contratación de médicos y toda licencia o autorización adquirida a virtud de leyes especiales anteriores y convalidadas por dicha ley al derogarlas.

ARTICULO 2.—Los médicos que a 1ro. de enero de 1957 estuvieren ejerciendo la profesión médica a virtud de la ley

especial para la contratación de médicos quedan sujetos a las siguientes disposiciones:

(1) Todo médico que, siendo ciudadano extranjero, hubiere tomado y aprobado un examen regular ofrecido por el Tribunal en convocatoria ordinaria de acuerdo con la ley del ejercicio de la profesión médica, en condiciones iguales a las que rigen para los que toman y aprueban dicho examen regular a los fines de la ley del ejercicio de la profesión médica, y que a virtud de su aprobación recibiera licencia provisional especial, tendrá derecho a que el Tribunal le otorgue, y dicho Tribunal le otorgará, la licencia permanente al someter a dicho Tribunal evidencia de haber adquirido la ciudadanía de Estados Unidos siempre que ésto lo haga en o antes de 30 de junio de 1961.

Disponiéndose: (a) Que el que hubiere tomado dicho examen una (1) vez con anterioridad a la aprobación de esta ley y no la hubiera aprobado en todo o en parte, tendrá —y le será dada por el Tribunal— una segunda y última oportunidad que será en la primera convocatoria ordinaria después de aprobada esta ley; si aprobase en su totalidad el examen regular le cobijarán entonces las disposiciones del primer párrafo de este artículo.

(b) Que, si hubiere aprobado dicho examen y/o exámenes, o hasta tanto tome y apruebe el segundo y hasta tanto adquiera la ciudadanía de Estados Unidos le sea extendida la licencia permanente, podrá seguir ejerciendo la medicina sobre la base de la licencia provisional especial y con sujeción a las condiciones que establece la ley especial para la contratación de médicos.

(2) Todo médico que hubiere obtenido o retenido licencia provisional especial sin examen regular de convocatoria ordinaria según se establece en el apartado 1 del Artículo 1, sea cual sea su ciudadanía para la fecha de vigencia de esta ley, deberá tomar el examen regular que el Tribunal ofrezca en la convocatoria ordinaria próxima inmediata a la vigencia de esta ley. La aprobación de este examen dará opción a licencia permanente la cual le será otorgada por el Tribunal si para entonces el médico así examinado y aprobado ha adquirido la ciudadanía de Estados Unidos siempre que ésto se haga no más tarde del 30 de junio de 1961.

Disponiéndose, (a) Que al que no aprobare en todo o en parte dicho examen le será dada una segunda y última oportunidad de tomar y aprobar en la convocatoria ordinaria siguiente todo el examen o la parte de él que hubiere dejado de aprobar. Si aprobare entonces en su totalidad el examen

regular, tendrá derecho a que el Tribunal le otorgue, y el Tribunal le otorgará, licencia permanente si es para ese entonces el médico así reexaminado y aprobado ciudadano de Estados Unidos o al recibir evidencia de que el médico así reexaminado y aprobado ha adquirido la ciudadanía de Estados Unidos siempre que ésto se haga no más tarde de 30 de junio de 1961.

(b) Que mientras toma dicho examen y reexamen y —de aprobarlo— mientras adquiere la ciudadanía de Estados Unidos y la licencia permanente podrá seguir ejerciendo la medicina sobre la base de la licencia provisional y los términos sujeto a los cuales le fué ésta concedida.

ARTICULO 3.—Todo médico comprendido en esta ley que deba tomar un examen o un reexamen total o parcial de acuerdo con las disposiciones de esta ley y no lo haga en la convocatoria ordinaria que le corresponde según aquí se establece habrá renunciado a todo derecho que por esta o cualquier otra ley se le confiera y su licencia provisional especial será nula a la fecha del comienzo de los exámenes regulares de esa convocatoria. Todo médico que, con opción a licencia permanente por haber aprobado el examen regular que aquí se establece, no haya obtenido la ciudadanía de Estados Unidos para la fecha que en esta ley se fija habrá perdido en esa fecha toda opción a licencia permanente; y en esa fecha será nula su licencia provisional especial.

ARTICULO 4.—La convicción de cualquier médico a quien esta ley cobije por cualquier delito **felony** será considerada causa suficiente para que no pueda de ahí en adelante acogerse a sus disposiciones.

ARTICULO 5.—Ninguna disposición o parte de esta ley habrá de entenderse o interpretarse en el sentido de que anula, enmienda o en forma alguna altera las disposiciones o partes de la ley del ejercicio de la profesión médica.

ARTICULO 6.—Esta ley, por ser de carácter urgente y necesario, empezará a regir inmediatamente después de su aprobación.

Lo creemos bueno y estamos convencidos de que habrá de encontrar fuerte objeción por parte de algunos oficiales municipales y de algunos legisladores. Preparémonos con tiempo a dar esta batalla.

Comité de Narcóticos

Convencidos de que el narcómano no recibe atención adecuada en nuestra comunidad y de que invariablemente se le tilda de

criminal en lugar de "enfermo", designamos un comité especial para que estuviera este problema. Oportunamente oiremos su informe y recomendaciones.

Comité de Relaciones Públicas

Los miembros de este comité han emprendido la árdua labor de acercarnos y darnos a conocer al pueblo, en forma valiente e inteligente. Consideramos este uno de los problemas más difíciles que nos confronta. Exhorto a todos a estudiar con detenimiento las recomendaciones de este comité.

Directorio Médico en la Prensa

Creemos que la forma sobria y concisa como está apareciendo el Directorio de esta Asociación se ajusta a la filosofía de esta Cámara. Implica más trabajo para nuestro personal administrativo pero esto se justifica ampliamente por el prestigio que recuperan nuestros anunciantes.

Sección de Médicos Generalistas

En medio de un gran entusiasmo logramos formar la Sección de Médicos Generalistas. Raras veces hemos visto un grupo de médicos tan entusiastas y sinceros. Merecen el respaldo de toda nuestra Asociación y el reconocimiento, como grupo, de nuestros hospitales para que se le brinden a ellos facilidades al hospitalizar sus pacientes. Más tarde consideraremos el reglamento de esta nueva sección.

Centro Médico

Esta Presidencia participó en las deliberaciones de dos de los subcomités del proyectado Centro Médico de Puerto Rico.

Esta Cámara de Delegados, en su última reunión de Agosto en Ponce, se expresó enfáticamente en contra de la creación de facilidades para pacientes privados en los hospitales del Centro Médico. Hemos celebrado reuniones con el Secretario de Salud y con el Decano de nuestra Escuela de Medicina y en forma clara y sin ambajes, les hemos informado de nuestros propósitos de no claudicar. El Dr. Henry Clark, asesor especial del comité timón del centro médico, ha recomendado favorablemente en su informe final con respecto a la creación de facilidades para pacientes privados en el Centro Médico. Esta Asociación no debe ceder, y debe continuar en forma valiente y serena en la presentación de sus puntos de vista.

Otro subcomité al cual pertenecemos es el llamado de Educación Médica. Este subcomité realizó un estudio comparando los conocimientos médicos de los graduados de universidades recono-

cidas con los graduados de universidades no reconocidas. Se usaron como medidas de comparación las clasificaciones obtenidas por los médicos durante su año de internado y las clasificaciones obtenidas en los exámenes de reválida. Invariablemente, los graduados de escuelas no reconocidas demostraron poseer una educación médica muy inferior a los graduados de universidades reconocidas. Se recomendó que todo solicitante a internado de una Universidad no reconocida debe someterse a un examen preliminar antes de que se le permita iniciar su internado; de no ser satisfactorio el resultado de ese examen el candidato se verá obligado a tomar por lo menos un año de preparación adicional en la Universidad de Puerto Rico.

Comité de Servicios Médicos

Encomendamos a este comité la realización de un estudio entre nuestra clase para determinar cuáles son los honorarios médicos usuales en Puerto Rico. Este estudio aún no se ha terminado y creemos debe continuarse.

Somos de opinión que el Comité de Servicios Médicos se debe convertir en uno de los más importantes de nuestra Asociación. Sugerimos que al igual que en la Asociación Médica Americana se le añadan los siguientes subcomités:

1. De servicios médicos federales.
2. De planes de servicios médicos voluntarios patrocinados por los médicos.
3. De planes de servicios médicos patrocinados por los suscriptores (cooperativismo).
4. De planes de servicios médicos de uniones de obreros.

Es bueno apuntar respecto a los dos últimos planes mencionados que la Asociación Médica Americana ha tomado posiciones claras y definitivas y que además ha sentado las bases sobre las cuales deben regirse esos planes. Es por todos sabido que en nuestro país hay ciertos sectores que están interesados en desarrollar programas de esa índole y nos atañe a nosotros el tratar de inmiscuirnos en la formación de los mismos, de suerte que sus normas resulten aceptables a nuestros conceptos de ética médica.

Comités de enlace con Farmacéuticos y Abogados

Nos hemos podido percatar que existen áreas de fricción en las relaciones de los médicos con los farmacéuticos y con los abogados. Hemos designado sendos comités que habrán de suavizar asperezas y de presentarnos recomendaciones en cuanto a la actitud que debemos asumir en nuestros contactos con estos profesionales.

Programa Medicare

Desde ayer, el 7 de diciembre de 1956, está oficialmente en vigor la ley 569 del 84avo. Congreso que autoriza la prestación de servicios médicos a los familiares hospitalizados del personal militar en servicio activo.

Esta Asociación ha firmado contrato con el Departamento de la Guerra comprometiéndose a actuar como agente administrativo en el programa local. La tabla de honorarios propuesta por nosotros ha sido aceptada con muy pocas y ligeras alteraciones. Para asesorarnos en el aspecto administrativo de Medicare, contamos con los valiosos servicios del Sr. Antonio Laloma, quien ya ha organizado nuestra oficina para hacerle frente a los nuevos deberes.

Este programa tiene una magna importancia, pues por vez primera el gobierno ha negociado con la medicina organizada en Puerto Rico para rendir servicios a un sector de la comunidad. Se hace mandatorio que todos cooperemos y lo que es más importante que actuemos de buena fe y en forma justa y razonable. Habrá un comité de nuestra Asociación que velará por el buen funcionamiento del programa y que aplicará sanciones en caso de fallas o abusos. Actuemos en forma correcta, ciñámonos al reglamento y el éxito será nuestro.

Junta de Auxilio Médico Mutuo

Esta Cámara encomendó a la Junta del Auxilio Médico Mutuo ciertos estudios actuariales con respecto a nuestro plan. Le ruego le presten atención a las recomendaciones de la Junta, las que presentarán en breve.

Plan de Regionalización

Vuestra Junta de Directores tuvo una reunión con el Hon. Secretario de Salud y su Subsecretario y después de una larga y franca discusión del plan de Regionalización conseguimos la promesa del Secretario de que los jefes de los servicios no funcionarán como consultantes gratuítos en casos de pacientes particulares como se había ideado originalmente y a lo cual esta Cámara de Delegados se opuso tan decididamente. Hemos seguido muy de cerca el desenvolvimiento del plan de regionalización y por ahora no hemos sido informados de otros aspectos del mismo que consideremos nocivos a nuestra clase.

Contrato Veteranos

Un comité especial de nuestra Asociación presidido por el Dr. Guillermo Picó, sostuvo varias entrevistas con el Director de la

Administración de Veteranos, Dr. Jaime Serra Chavarry, tendientes a conseguir que se cambiaran las normas establecidas por la Administración en los contratos de hospitalización. Las gestiones realizadas tuvieron éxito y con fecha 13 de agosto recibimos la siguiente carta del doctor Serra Chavarry:

August 13, 1956

Dr. Jaime F. Pou
President
P. R. Medical Association
Santurce, P. R.

Dear Dr. Pou:

This is in answer to your letter of July 26, 1956.

It is in order to state that, although we have not received a reply to our communication from our Area and Central Offices regarding your wishes as stated in above mentioned letter, we are in agreement with your petition, and we also assume that Central Office will allow us to go ahead with the addendum to the contract.

As of today we are informing the contract hospitals to secure the signatures of their respective staffs to the statement, and I quote you, "that physicians who are to render the services to the contract hospital will ratify the table of fees and consent to carry out the services for the stipulated amounts."

Our first letter to this effect is being mailed to the Presbyterian Hospital, Santurce, P. R. This type of letter will be sent to **"all"** contract hospitals no matter what type of contract they have. As to the other type of contract (sic) we are notifying them that no renovation will be accepted unless it conforms to the above agreement. As you know the renewals of contracts of this type are made every 6 months, and most of them become due during September and October, 1956.

We hope that this arrangement, in which we have acquiesced to the wishes of the Puerto Rico Medical Association is entirely satisfactory. We are sure of the cooperation of the Association with the Veterans Administration and you may be sure of the wishes of the VA to maintain the best relations with your society.

Very truly yours,

J. SERRA CHAVARRY, M.D.
Manager

La Asociación Médica Americana

Debemos estar verdaderamente agradecidos a la Asociación Médica Americana por la ayuda tan importante que nos han prestado en forma desinteresada. En numerosas ocasiones hemos solicitado su ayuda y siempre han respondido a nuestra llamada. Nuestro Comité de Relaciones Públicas tuvo la oportunidad de ser asesorado por el Sr. Leo Brown, Oficial de Relaciones Públicas de la Asociación Médica Americana, quien dedicó cuatro días de su tiempo, sin costo alguno para nosotros, a explicarnos sobre la organización y funcionamiento de las actividades de relaciones públicas de una asociación médica. Igualmente útil fué la ayuda que nos prestara la Asociación Médica Americana con respecto al programa de Medicare.

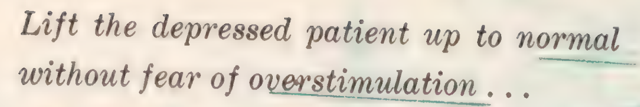
Las Relaciones de nuestra Asociación con las Esferas Gubernamentales

Han sido verdaderamente cordiales nuestras relaciones con todas las esferas del Gobierno local. Nuestros consejos y nuestros servicios han sido requeridos varias veces por la rama legislativa; la rama ejecutiva, incluyendo al Gobernador, la Secretaría de Estado, Salud y Obras Públicas nos han oído y han mostrado interés en nuestros problemas. Exhortamos a todos nuestros compañeros a que contribuyan en la medida que esté a su alcance a estrechar estos lazos y a cimentar estas relaciones. Creemos firmemente que si asumimos una actitud de cooperación y si ofrecemos ayuda positiva habremos de continuar en nuestra cadena de logros.

Nuestro más sincero agradecimiento a esta Honorable Cámara de Delegados por la ayuda que nos han brindado durante el año que acaba de pasar. La Junta de Directores que nos acompañó y nos estimuló en nuestra labor tendrán nuestro eterno agradecimiento.

JAIME F. POU, M.D.
Presidente





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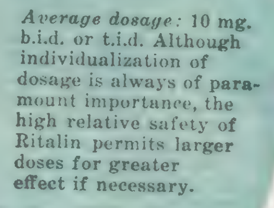


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Reference: 1. Pocock, D. G.: Personal communication.



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BOLETIN

DE LA

ASOCIACION MEDICA DE PUERTO RICO

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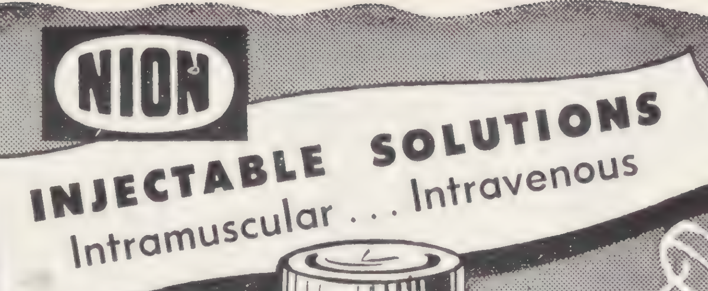
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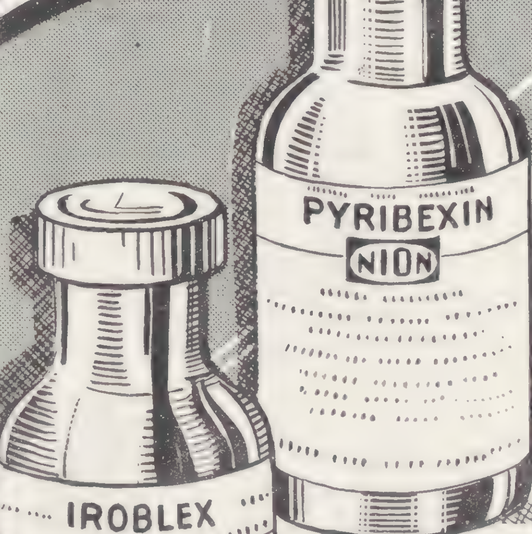
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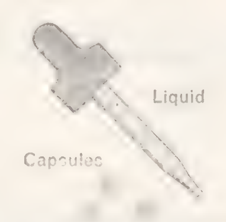
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COLACE Capsules, 50 mg., bottles of 30. COLACE Liquid (1% Solution) 30-cc. bottles with calibrated dropper.

(1) Wilson, J. L., and Dickinson, D. G.: J. A. M. A. 158: 261, 1955.



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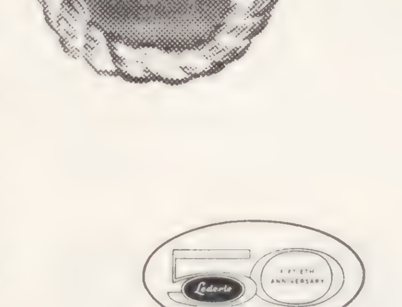


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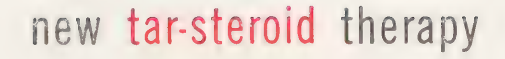
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> Recurrent episodes of various dermatoses "which became, eventually, refractory to hydrocortisone when used alone, occurred less frequently and did not become refractory to therapy" when the steroid was combined with an agent such as tar.[2]

1. Bleiberg, J.: Hydrocortisone-tar extract cream in chronic and sub-acute dermatoses, to be published.
2. Welsh, A.L., and Ede, M.: Hydrocortisone ointments: their rational use in dermatology, Ohio State M.J. *50*:837 (Sept.) 1954.

available: 7 gram and 1 ounce tubes.

formula: 0.5% hydrocortisone; 5% special coal tar extract (Tarbonis®)—greaseless, stainless hydrophilic vanishing cream base.

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Van Nuys, California



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More than fifty years of scientific cattle breeding at Carnation Farms have resulted in many famous Holstein champions. Cattle from these famous bloodlines are shipped to dairy farms across the country to help improve the Carnation milk supply.

2. CARNATION RESEARCH LABORATORY

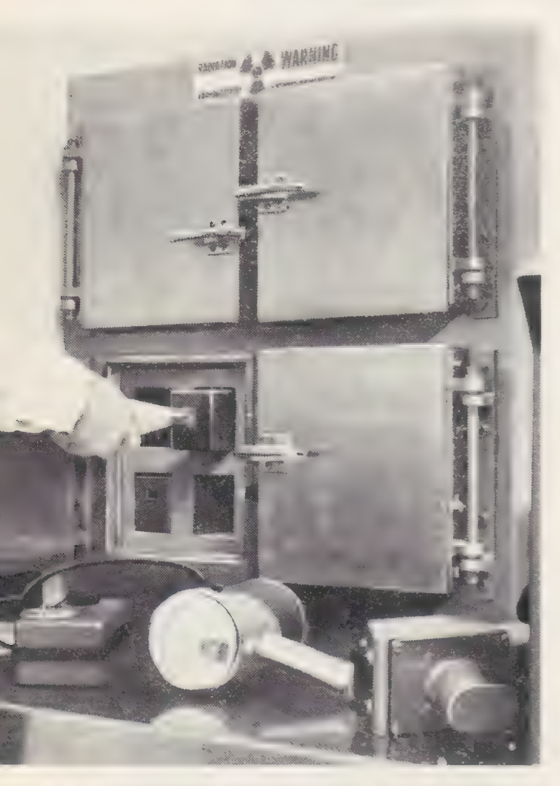
Newest research facility is the Carnation Research Laboratory at Van Nuys, California, shown above – one of America's most modern laboratories devoted to product research. In addition to direct research in dairy and cereal foods, Carnation Laboratory coordinates the other phases of Carnation Research.

3. QUALITY CONTROL RESEARCH

Laboratories at each Carnation Plant exercise rigid day-by-day control of Carnation Milk processing. Samples from *all* Carnation plants are rechecked at Central Product Control, Oconomowoc, Wisconsin, to assure *uniform* high quality of Carnation Milk everywhere.

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As a member of the National Research Council, Evaporated Milk Association, National Dairy Council, American Dry Milk Institute and many other similar organizations throughout the world, Carnation contributes importantly to the broad research activities conducted by these groups.



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Carnation sponsors University research in highly specialized fields, such as radiation and supersonic vibration, as related to dairy foods. Current projects are under way at the California and Massachusetts Institutes of Technology and the Universities of Wisconsin and Illinois.

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antibiosis intestinal aumentada



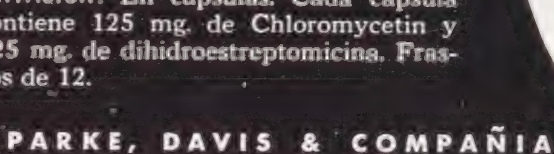
INDICACIONES: Infecciones entéricas de tipo diarreico
Infecciones mixtas en cirugía intestinal
Tuberculosis anorrectal
Fístula anal

PREOPERATORIAMENTE
Cirugía colónica y rectal

POSTOPERATORIAMENTE
Extirpación de quistes pilonidales.

DOSIFICACION: En casos de diarrea o en la enteritis disentérica, 2 a 4 cápsulas cada seis horas. En el preoperatorio de la cirugía intestinal, 2 a 4 cápsulas cada seis horas, durante los tres o cuatro días que preceden a la operación. Esta asociación antibiótica es particularmente útil en el postoperatorio, conjuntamente con la administración oral de líquidos. En los pacientes postoperatorios con infecciones causadas por patógenos comunes, este tratamiento debe continuarse durante cinco o seis días. Cuando se emplea en casos de fístula anal tuberculosa, el CHLOROSTREP debe ser administrado en dosis más elevadas o durante más tiempo.

PRESENTACION: En cápsulas. Cada cápsula contiene 125 mg. de Chloromycetin y 125 mg. de dihidroestreptomicina. Frascos de 12.



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La abundante proteína en Bíolac asegura el sano desarrollo de los tejidos del cuerpo, mientras que su reducido nivel de grasa facilita la digestión. Se le ha agregado lactosa para proveer un contenido adecuado de carbohidrato, y las vitaminas y hierro en Bíolac proporcionan satisfacción *completa* a las necesidades nutritivas.

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ganglionar autónomo

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La respuesta clínica es *predecible* y *reproducible* con exactitud no igualada por ningún otro agente hipotensivo.

Por vía oral *su absorción es casi de cien por cien.* Las demás drogas, bloqueadoras del sistema ganglionar, son escasamente absorbidas... generalmente menos del 20 por ciento.

Su acción hipotensiva comienza *gradualmente y dura de 6 a 12 horas.*

Es eficaz con frecuencia en *pacientes refractarios a todos los demás agentes hipotensivos.*



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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 48 DICIEMBRE, 1956 No. 12

ESTUDIO SOBRE LA POLIOMIELITIS AGUDA EN PUERTO RICO DURANTE EL PRIMER SEMESTRE DEL 1956

*ENRIQUE L. MATTA JR., M.D., F.A.A.P.**

Con el fin de ofrecer una orientación clara y al día sobre la incidencia de poliomielitis aguda registrada en la isla durante este año, así como los últimos informes sobre la Vacuna Salk, hemos preparado este trabajo para vuestra orientación y consideración.

Hemos creído conveniente examinar la data existente sobre los casos de poliomielitis aguda registrados en la isla durante el 1956 a la luz de dos consideraciones principales.

Primero: El hecho de haber pasado la población de esta isla por un brote epidémico de carácter severo y general que comenzó en noviembre de 1954, y terminó en julio de 1955.

Segundo: El hecho de haberse llevado a efecto con éxito un programa de vacunación contra la parálisis infantil en las edades menores de 10 años.

RESUMEN DEL BROTE EPIDEMICO DE NOVIEMBRE 1954 A JULIO 1955

1. Total de municipalidades en Puerto Rico ---------- 76
2. Total de municipalidades afectadas -------------- 63
3. Municipalidades exentas del impacto de polio durante este brote -- 13

Aguas Buenas	Hormigueros	Orocovis
Arroyo	Lajas	Rincón
Ceiba	Las Marías	Sábana Grande
Culebra	Maricao	Salinas
	Guánica	

* Consultor en Pediatría, Departamento de Salud, Estado Libre Asociado de Puerto Rico.

4. Total de casos registrados en 1955 434
5. Tipo paralítico (84.8%) 368
6. Distribución por zonas:
   a. Urbanas ....(29.0%)............ 126
   b. Rural ....(71.0%)............ 307

Cifras de 1940-1954 presentan la incidencia de polio en un 44% de casos urbanos y 56% de casos rurales — se deja ver en el brote de 1955 una deviación hacia una mayor incidencia en la población rural.

7. Total de defunciones 19
8. Total de casos registrados durante el brote 1954-1955 517
9. Total de defunciones registradas durante el brote de 1954-1955 25
10. Coeficiente de mortalidad 4.8%

RESUMEN DE LA VACUNACION SALK A JULIO 1956

1. Grupo primero — 6 meses a 2 años ...... (187,000 niños)

Primera dosis	88,198
Segunda dosis	72,092
Tercera dosis	32,625
Total de dosis aplicadas	192,915

2. Grupo segundo — 3 - 5 años ...... (232,000 niños)

Primera dosis	65,733
Segunda dosis	58,608
Tercera dosis	25,357
Total de dosis aplicadas	149,698

3. Tercer grupo — 6 - 9 años ...... (255,000 niños)

Primera dosis	223,553
Segunda dosis	200,481
Tercera dosis	30,589
Total de dosis aplicadas	454,423

4. Cuarto grupo — 10 - 15 años:

Primera dosis	53,564
Segunda dosis	48,691
Tercera dosis	1,271
Total de dosis aplicadas	103,526

5. Niños de edad infantil y preescolar (6 meses a 5 años)

Total de niños que recibieron la primera dosis	153,931
Total de niños que recibieron segunda dosis	130,700
Total de niños que recibieron tercera dosis	57,982
Total de dosis administradas	342,613

6. Niños de edad escolar (6 años a 15 años)

Total de niños que recibieron primera dosis	276,917
Total de niños que recibieron segunda dosis	249,172
Total de niños que recibieron tercera dosis	31,860
Total de dosis administradas	557,949

7. Total de niños vacunados con una dosis 430,828
8. Total de dosis de vacuna Salk administradas 900,562
9. Reacciones reportadas de carácter simple y pasajero, tales como: **Fiebre, malestar general, urticaria.**

RESUMEN DE LOS CASOS DE POLIOMIELITIS REGISTRADOS EN PUERTO RICO DURANTE EL PERIODO DE ENERO A JULIO 1956

1. Total de casos registrados y confirmados 32

2. **Tipo de poliomielitis —**

Paralítico	(84.4%)	27
No paralítico	(15.6%)	5

3. Muertes (niña de 10 años, señora de 42 años) ... 2

4. **Poblaciones que han reportado casos** 18

Comerío	5	San Juan	1
Guayama	2	Aguadilla	1
Santurce	4	Carolina	2
Utuado	1	Guaynabo	1
Río Piedras	4	Bayamón	1
Cataño	1	Guayanilla	1
Caguas	2	Cayey	1
Juncos	1	Lares	1
Morovis	1	Naguabo	1
Ponce	1		

5. **Incidencia por mes** — estudio comparativo:

Mes	1954	1955	1956
Enero	7	116	1
Febrero	6	85	1
Marzo	1	77	2
Abril	1	58	2
Mayo	1	38	8
Junio	0	28	10
Julio	0	17	8

(1954 se considera un año con incidencia normal en estos meses comparados)

6. **Distribución de casos por edades:**

Primer grupo - 16 casos (6 meses a 2 años) ______ 16
- 5 meses ______ 1
- 6 meses ______ 1
- 7 meses ______ 1
- 8 meses ______ 4
- 10 meses ______ 1
- 1 año ______ 2
- 2 años ______ 5
- 2½ años ______ 1

Segundo grupo (3 - 5 años) ______ 7
- 3 años ______ 3
- 3½ años ______ 1
- 4 años ______ 3

Tercer grupo (6 - 9 años) ______ 5
- 6 años ______ 1
- 7 años ______ 1
- 10 años ______ 3

Cuarto grupo (mayores 10 años) ______ 4
- 12 años ______ 1
- 16 años ______ 1
- 24 años ______ 1
- 42 años ______ 1

7. **Distribución comparativa de casos en por ciento del total:**

	1955	1956	1955	Cum.	1956	Cum.
Menores de 6 meses ____	11	1	3.0	3.0	3.1	3.1
6 - 11 meses ____	49	7	13.5	16.2	21.9	25.0
1 - 2 años ____	193	8	53.2	69.7	25.0	50.0
3 - 5 años ____	71	7	19.6	89.3	21.9	71.9
6 - 9 años ____	27	2	7.6	96.9	6.2	78.1
10 años en adelante ____	12	7	3.1	100.0	21.9	100.0

8. **Distribución por zonas:**

	1955	1956
Urbana	29%	60%
Rural	71%	40%

9. **Relación a Vacuna Salk — por edades:**

Total de niños vacunados y reportados luego con polio — 4
4 años — 1 caso
10 años — 3 casos
Todos recibieron dos dosis de vacuna Salk.

10. **Características de los casos vacunados que desarrollaron síntomas de poliomielitis aguda:**

a. Sintomatología en estos casos vacunados muy atípica.

1) Fiebre - presente en todos los casos
2) Dolor de cabeza - un caso
3) Rigidez nucal - tres casos
4) Dolor lumbo-toráxico - ningún caso
5) Dolor de garganta - dos casos
6) Vómitos - ningún caso
7) Diarrea - ningún caso
8) Estreñimiento - dos casos
9) Dolor muscular - tres casos

b. Los reflejos patelares, presentes en dos casos, ausentes en dos casos.

c. Líquido céfalo-raquídeo

Células: 1 caso con 20 células
2 casos sin células
1 caso sin examen

Proteina: 40 mg., 20 mg., 52 mg.
1 caso sin hacerse este examen

d. Tipo de parálisis

Parálisis caracterizada por ser de carácter leve y con rápida mejoría.

Parálisis espinal - 3 casos, secuela de menor importancia.

Parálisis bulbo espinal - 1 caso de carácter severo.

e. Coeficiente de mortalidad - 62% - un aumento sobre el coeficiente de mortalidad de 4.8% reportado durante el brote epidémico 1954 - 55.

11. **Observaciones generales sobre la poliomielitis aguda en Puerto Rico según estudio de los casos registrados durante los primeros siete meses del 1956.**

a. Cambio radical en la incidencia mensual, observándose un aumento marcado este año en el mes de mayo, continuado a junio y julio. Esta tendencia es similar a la observada en los Estados Unidos y contraria a la observada hasta ahora en Puerto Rico.

b. Mayor número de casos en la zona urbana deja ver la corrección de la deviación observada durante el brote.

c. Cambio en las edades afectadas observándose **una reducción** en las edades 1 - 2 años y 6 - 9 años, un leve aumento en las edades de 6 - 11 meses y 3 - 5 años, y un aumento marcado en las edades mayores de 10 años y adultos.

d. Las dos muertes ocurrieron en mayores de 10 años.

e. **Incidencia de casos de poliomielitis**

		Casos			Tasa por 100,000		
	Pob.	P	NP	Total	P	NP	Total
Total vacunados	430,828	4	0	4	0.9	0	0.9
Total no vacunados	296,516	21	3	24	7.0	1.0	8.0

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4. Alonso Ricardo, Jefe, Departamento de Pediatría, Hospital de Distrito de Fajardo (comunicación personal).

5. Mendoza P., Jefe, Departamento de Pediatría, Hospital Distrito Aguadilla (comunicación personal).

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II. LA POLIOMIELITIS Y LA VACUNA SALK

Hemos revisado la literatura recientemente publicada sobre la Poliomielitis y la Vacuna Salk, con el propósito de extractar aquella información de interés general, tanto para el médico en el ejercicio privado de la profesión, como el de Salud Pública.

Queremos mencionar los siguientes trabajos de los cuales hemos extractado la mayor parte de la información que discutiremos a continuación.

1. "Seminar" presentado ante la Academia Americana de Pediatría en su reunión de octubre 1955 sobre "Recent Development in Infectious Diseases" y un resumen del cual se publicó en la revista médica "Pediatrics" en junio 1956.
2. Symposium presentado ante la Sociedad Médica del Condado de Nueva York en su reunión de marzo 1956, sobre "Nuevos Horizontes en Poliomielitis" publicado en la revista médica, New York Medicine en julio 1956.
3. Manual intitulado "New Information for Physicians on the Salk Poliomyelitis Vaccine" recopilado por Dr. Hart E. Van Riper, Director Médico de la Asociación Nacional de la Parálisis Infantil, publicado en junio 1956.

Con el fin de facilitar la claridad e interés de esta información, la presentaremos en forma de preguntas y respuestas.

LA POLIOMIELITIS

1. Cómo y por dónde entra al cuerpo el virus del polio?

a. El virus no se propaga por insectos que atacan al hombre y sí se propaga principalmente de persona a persona.
b. Puede entrar por las siguientes vías:
   1. Olfatoria
   2. Faríngea
   3. Oral

2. ¿Cuál es el sitio de la lesión inicial?

a. Según Bodian, en ciertos tejidos desconocidos de la parte superior e inferior del conducto gastrointestinal. Relación de eventos: invasión por conducto gastrointestinal, multiplicación del virus, viremia, invasión secundaria de los ganglios con formación de lesiones.
d. Según Faber, en los ganglios periferales, que suplen el ca-

nal gastrointestinal, especialmente en la parte superior, poco después de haber sido expuesto a la enfermedad.

c. La faringe es más vulnerable a la infección que la parte inferior del conducto gastrointestinal.

3. **¿Cómo se elimina el virus?**

a. El virus se elimina de la garganta y el intestino, por varios días antes de la aparición de síntomas, llega a su máximo durante la fase aguda, y persiste reduciéndose gradualmente por varias semanas. Hay evidencia de eliminación faríngea e intestinal que se origina en los ganglios periferales que suplen el conducto gastrointestinal. Por lo tanto, la eliminación puede ser interna de focos neurales, así como de multiplicación viral en la mucosa intestinal (Faber).

b. Al eliminarse mayores y mayores cantidades de virus, se estimulan las defensas inmunológicas resultando importante secuela en cuanto a la extensión de la infección y la producción de la viremia.

4. **¿Cómo se produce la viremia?**

a. El mecanismo exacto se desconoce.

b. Según Faber, la viremia empieza tan pronto las fases de la infección han llegado a tal punto que se están eliminando grandes cantidades de virus al lumen del conducto alimenticio.

5. **¿Qué mecanismos son responsables de la invasión del sistema nervioso central?**

a. Invasión vía los nervios, más común en la parálisis de tipo bulbar.

b. Invasión vía la viremia, pasando directamente de la corriente sanguínea a la célula nerviosa, causa más frecuentemente manifestaciones espinales con parálisis. (Faber).

6. **¿Cuál es la patogenia de las infecciones simples y asintomáticas?**

Casos leves pero sin parálisis frecuentemente se deben a infección del sistema nervioso central que cede antes de producir daño irremediable.

7. **¿Cuáles son las defensas naturales y adquiridas contra la infección poliomielítica, tanto en su aspecto primario como secundario?**

   a. Si no tiene cierto grado de inmunidad, el individuo tiene poca o ninguna defensa contra la infección primaria, y desarrolla la enfermedad asintomática o levemente sintomática.
      Las razones para esto pueden estar en el cuerpo del paciente, o deberse a la cepa del virus invasor.
   b. Los individuos con cierto grado de inmunidad pero aún susceptibles, tienen grandes probabilidades de no desarrollar la enfermedad, o desarrollarla con carácter leve.
   c. Los anticuerpos en el moco faríngeo, según han demostrado Bell y otros, pueden neutralizar virus ingerido presente en la faringe, y de esa forma evitar la entrada del virus en este punto. No hay anticuerpos en el intestino.
   d. Si el individuo adquiere una infección, los anticuerpos en la sangre, a un nivel adecuado pueden neutralizar el virus que circula y evitar la invasión virémica del sistema nervioso central.
   e. Inmunidad humoral es poco efectiva en detener la invasión neural. Sin embargo, esta inmunidad humoral u otro factor desconocido aparentemente puede detener hasta cierto punto el avance de la infección después de haber sido invadido el tejido neural. Esto evita o aminora los efectos paralíticos de la invasión del sistema nervioso central.
   f. En resumen podemos decir que el caso asintomático es uno en el que la infección se limita a los ganglios periferales, o en algunos casos donde el sistema nervioso central ha sido afectado levemente sin producir manifestaciones clínicas.
   g. En sus formas sintomáticas, la infección ha pasado al sistema nervioso central por vía neural o sanguínea a grado tal que produce ciertas manifestaciones.
      1. En los casos leves no paralíticos—los motoneurones están poco afectados.
      2. En casos leves con parálisis—hay motoneurones afectados en menor grado, y a menudo de carácter temporero.
      3. En los casos severos de parálisis—los motoneurones, en parte por lo menos, han sido afectados irremediablemente.

8. **¿Qué justificación hay para vacunar activamente la población contra la poliomielitis?**

   a. Estudios hechos revelan que casi ninguno de los niños de 3 a 5 años tienen anticuerpos contra los tres tipos virus

poliomielíticos, y menos del 40% de este grupo tiene anticuerpos contra el tipo 1. De hecho solamente el 50% de las personas mayores de 18 años de edad tienen anticuerpos contra los tres tipos de virus.

b. Anticuerpos contra un tipo de virus no asegura protección contra los otros dos tipos de virus.

c. En Puerto Rico el ritmo de progreso general socio-económico mejorando las condiciones de vida ha demostrado plenamente la característica principal de la poliomielitis, a saber, el ser ésta una enfermedad de los países más civilizados al alejarse el individuo de la fuente de contagio e inmunización natural, como lo es la excreta humana. El continuado progreso general de nuestra isla nos haría esperar epidemias más frecuentes y más severas.

9. ¿Qué posibilidades hay de una prueba diagnóstica de poliomielitis?

a. No se anticipa prueba alguna al presente que ayude al diagnóstico de polio antes de que aparezca la parálisis.

b. No hay forma de identificar un caso de polio con invasión del sistema nervioso antes de aparecer la parálisis.

c. No hay forma de identificar el caso de polio con leves síntomas, similares al de otras enfermedades, a tiempo suficiente para ser de importancia.

d. Pruebas en casos diagnosticados de poliomielitis:

1) El virus puede aislarse e identificarse en menos de una semana.

2) La prueba de fijación de complemento es positiva al comienzo de la enfermedad, y los resultados pueden leerse en 24 horas.

3) Prueba comparativa de los niveles de anticuerpos observados a tres semanas de diferencia.

4) Las pruebas de diagnóstico pueden ser de gran utilidad en la identificación de los casos atípicos de polio y en el diagnóstico diferencial.

10. ¿Por qué deberemos preguntarnos cuando veamos un caso de parálisis, si es polio o no?

a. Porque casos clínicamente típicos de poliomielitis, pueden haber sido causados por otros agentes o un tipo 4 de polio aún no identificado. Dr. Linnet del Laboratorio Viral de California analizó 69 casos típicos de parálisis aislando viruses en todos, menos 18 casos. De los 18 casos, solamente

13 dieron evidencia serológica de tener poliomielitis, dejando 5 casos de poliomielitis paralítica típica en los cuales no había evidencia de laboratorio de que habían tenido polio. Otros laboratorios han tenido experiencias similares.

b. Hay ciertos casos que no están protegidos por la vacuna actual. Puede descubrirse un cuarto tipo de polio o un virus que cause parálisis que no reconocemos al presente.

11. **¿Hay alguna droga efectiva contra el virus de polio?**

a. Ninguna al presente.
b. No hay forma de evitar que el paciente con polio sea un portador del virus en su excreta.
c. Los Laboratorios Eli Lilly tienen la única droga bajo estudio, conocida por 8450, que tiene el poder de inmunizar un animal por pocos días contra virus virulentos, mayormente neurotrópicos, y entre ellos, el de polio.
Se está probando la efectividad de esta droga durante la fase virémica antes de que el virus invada el sistema nervioso y en la fase intracelular de la infección.

12. **Uso presente de la Gamma Globulina en la prevención de polio.**

a. El uso de la Gamma Globulina no es una medida práctica de salud pública.
b. Puede usarse en el caso de un contacto directo de corta duración con un paciente durante la fase aguda de su enfermedad—o sea, si el niño ha sido expuesto **por un día**, a una persona que **un día más tarde** presenta síntomas de la enfermedad, siempre y cuando el médico vea al contacto **dentro de los 2 a 3 días siguientes** de haber sido expuesto a la enfermedad.
c. En una familia donde ha surgido un caso de polio no está indicado vacunar a los contactos, ya que el diagnóstico se hace al tercero o cuarto día de fiebre, o sea, al 5to. ó 6to. día de tener el virus en la nariz y garganta. El período de incubación, siendo de 7 a 9 días, y la familia habiendo estado expuesta por 4 a 5 días, no hay indicación para el uso de Gamma Globulina.

13. **¿Qué debemos saber sobre el tratamiento de poliomielitis?**

a. No tenemos tratamiento alguno.
b. Sólo hacemos tratamiento sintomático y de las complicaciones.

c. En lo que hemos mejorado grandemente es en el tratamiento de pacientes con formas severas de la enfermedad, tal como la poliomielitis bulbar y la parálisis de los músculos respiratorios. El éxito se ha debido a los Centros Respiratorios establecidos y equipados por la Fundación Nacional Contra la Parálisis Infantil, y la inclusión del fisiólogo como recurso del grupo médico.
d. Se ha mejorado la rehabilitación de funciones musculares de estos pacientes.
e. Se ha mejorado el tratamiento de los defectos circulatorios que se presentaban en los casos de polio bulbar y de las complicaciones renales.
f. Daño renal y cor pulmonale serán menos común como complicaciones de esta enfermedad.
g. El uso del peto respiratorio y la cama oscilatoria han libertado al paciente del tanque de respiración artificial.

14. **¿Qué condiciones deberemos considerar en el diagnóstico diferencial de poliomielitis aguda?**

a. Guillain Barre (Poliradiculoneuritis) P R N
   1) En la etapa inicial un contaje celular normal y una proteína elevada en el líquido céfalo-raquídeo son características de esta enfermedad.
   2) El período de invasión que es constante y febril en polio no existe en Guillain Barré, ya que éste se presenta desde el principio con parálisis sin síntomas premonitores, fase de invasión o fiebre.
   3) La aparición de la parálisis que es abrupta y de súbito en polio, es a veces gradual en Guillain Barré.
   4) La fase durante la cual la parálisis se extiende es de corta duración en polio, no habiendo peligro de mayor complicación muscular después del tercer día. En Guillain Barré por el contrario, la parálisis se extiende a toda una extremidad con bilateralidad y simetría marcada por muchos días.
   5) La topografía de la parálisis es diferente en ambos casos. La de polio se distribuye caprichosamente sin orden ni simetría. En Guillain Barré la parálisis se extiende por una extremidad con bilateralidad y simetría.
   6) En el período inicial la polio hace un ataque masivo de las funciones motoras, y diferentes músculos se afectan de diversas formas.
   En Guillain Barré el ataque es más limitado relativamente, siendo más en forma de una paresis pronuncia-

da que parálisis, a la vez que se distribuye al igual en todos los músculos en la región afectada.

7) Los reflejos quedan abolidos en la región paralizada en polio, (excepto en casos inmunizados). En Guillain Barré, al contrario, hay una abolición difusa de todos los reflejos tendóneos aún en áreas aparentemente normales.

8) Dolores espontáneos más agudos en polio; Guillain Barré se caracteriza por parestesias de las extremidades.

9) Hay cambios sensores presentes en Guillain Barré.

10) Líquido Céfalo-Raquídeo—pura disociación albúmino-citológica sin cambio en el contaje celular presente en Guillain Barré. La hiperalbuminosis aparece temprano, queda de manifiesto al cuarto día de enfermedad, llega a máxima intensidad al séptimo día, y persiste por varias semanas.

11) El curso de ambas enfermedades es distinto.—
En polio la secuela con parálisis debe temerse.
En Guillain Barré puede esperarse restablecimiento completo sin parálisis.

b. Trauma a las extremidades con limitación de movimiento: Ej.: Fracturas, dislocación, separación epifisial.
c. Sinovitis no específica: De la cadera.
d. Osteomielitis aguda.
e. Adenitis cervical con o sin tonsilitis.
f. Fiebre reumática aguda.
g. Escorbuto.
h. Neuritis después de inyecciones intramusculares.
i. Histeria.
j. Parálisis de gengibre (triorthocresyl phosphate).
k. Dermatomiositis.
l. Hemiplegia secundaria a lesión cerebral.
m. Chorea paralítica.
n. Neuromielitis óptica aguda.
o. Parálisis periódica familiar.
p. Ataxia aguda en niños de origen cerebelar.
q. Mielitis aguda.
r. Meningoradiculomielitis.
s. Hematomielia traumática.
t. Siringomielia.
u. Polineuritis debida a:
   1) Difteria.
   2) Botulismo.
   3) Envenenamiento con arsénico, plomo, apiol, triorthocresyl phosphate.

LA VACUNA SALK

1. **¿Qué clases de vacunas hay al presente contra enfermedades de etiología viral?**

   Dos clases existen al presente:
   a. La vacuna del virus muerto —ésta induce inmunidad a pesar de consistir del virus no infeccioso luego de tratamiento físico (rayos ultra violeta) o químicos (formaldehido, fenol, etc.)
   b. Vacuna de virus vivo—contiene mutantes que producen una infección modificada pero producen inmunidad contra la enfermedad viral original, ejemplos del cual son la vacuna contra la fiebre amarilla y la viruela.

2. **¿Qué clase de vacuna es la Salk?**

   a. La vacuna Salk en uso al presente es de la clase de virus muerto, eliminándose su poder infeccioso mediante tratamiento con formalina.

3. **¿Qué efecto tiene la Vacuna Salk en la producción de anticuerpos?**

   a. En personas sin anticuerpos al recibir la vacuna, produce un titraje intermedio de 1:16 a las dos semanas.
   b. La segunda inyección produce un titraje intermedio de 1:128.
   c. Un por ciento bajo de personas sin anticuerpos no desarrollan elevación alguna en titraje después de la inmunización.
   d. La dosis de reactivación (booster) no produce elevación alguna adicional antes de los 7 meses de la segunda inyección.

4. **¿Cuán efectiva es la Vacuna Salk?**

   a. Protección marcada contra la fase paralítica y particularmente, contra las formas bulbar y bulbo-espinal.

5. **¿Qué seguridad ofrece la Vacuna Salk?**

   a. Durante las pruebas de 1954 ningún caso de polio fué producido por la vacuna.
   b. Durante la primavera del 1955 algunos casos de polio fue-

ron causados por la vacunación, y otros casos secundarios resultaron de éstos.

c. Esta contaminación se debió a diferencias en rapidez del proceso de inactividad por la solución de formalina (1:4000) de partículas individuales en la misma solución.

d. Se han mejorado los procesos de filtración para eliminar partículas que puedan contener virus vivo.

e. Se han introducido nuevas pruebas y modificaciones de las pruebas con monos.

f. Desde la revisión de las pruebas de seguridad en mayo 1955, no hay evidencia de que lote alguno haya resultado contaminado.

Observación epidemiológica constante de cada caso de polio reportado en la nación ha sido mantenida constantemente.

6. **Cómo se garantiza la seguridad de la Vacuna Salk al presente?**

a. Mil cc de cada lote del tipo individual de cada vacuna se prueban repetidas veces para la presencia de virus vivo en cultivo de tejido.

b. Los lotes de los tres tipos de virus se mezclan y 1500 cc de esta mezcla trivalente son también probados repetidas veces en cultivo de tejido.

c. Muestras al azar de los frascos finales de cada lote son sometidos a prueba en cultivo de tejido y por inyecciones intra-cerebrales e intra-espinal en monos que han sido tratados con dosis altas de cortisona o irradiación del cuerpo entero para hacerlos más susceptibles al virus de la poliomielitis.

7. **Puede la inyección de reactivación (booster) de los 7 meses, aplicada después de la primera dosis, inducir una protección tan efectiva como si hubiere sido después de la segunda inyección?**

a. Puede ser tan efectiva como si fuera después de la segunda.

b. No se sabe aún a qué tiempo es mejor aplicar la dosis de reactivación, si a los 7, 10 ó 14 meses después de la segunda inyección. La primera dosis efectiva es una dosis de sensibilización. El efecto de tal inyección no se pierde si la segunda vacuna se tarda de 6 meses a un año o más. Si la segunda dosis se da de 6 a 12 meses actúa como una dosis de reactivación (booster).

La segunda inyección aplicada de 2 a 6 semanas no actúa como una dosis de reactivación (booster) por ser un lapso de tiempo demasiado corto para permitir una verdadera

elevación de anticuerpos en el 80% de los niños que se sensibilizaron con la primera dosis. En el restante 20% actúa como la primera dosis. Por lo tanto, la tercera dosis debe considerarse como una dosis de reactivación para todos.

8. **Qué acción se debe tomar si la dosis de reactivación de los 7 meses concurre con prevalencia de poliomielitis en cierta área?**

   a. Se debe proseguir con la vacunación de los que han recibido las dos primeras inyecciones así como empezar la vacunación de los que no la han recibido aún.
   b. No hay evidencia definitiva sobre el efecto provocativo de la vacuna.

9. **¿Están disponibles algunos resultados preliminares del programa de vacunación del 1955?**

Area	Tasa de Parálisis por 100,000		Tasa de no paralíticos por 100,000	
	Vacunados	Sin vacunar	Vacunados	No vacunados
California	3.3	10.0	11.9	10.4
Connecticut	5.7	20.1	35.8	66.0
Florida	1.3	4.9	15.4	11.1
Georgia	3.4	7.6	3.4	7.2
Illinois	1.4	10.4	12.6	24.5
Maryland	3.6	17.1	--	--
Minnesota	2.7	30.1	18.7	36.1
New York City	5.4	21.8	7.8	36.8
New York State	4.0	20.9	28.5	39.4
North Carolina	2.0	10.8	9.7	25.0
Oregon	2.1	15.2	4.2	8.7
Washington	5.8	21.0	1.4	10.5
Totales	3.2	13.2	14.7	19.7
Puerto Rico	0.9	7.0	0.0	0.0

10. **¿Qué reacciones a la vacuna han sido reportadas?**

   a. Reacción febril, reacciones locales y manifestaciones alérgicas.
   b. La cantidad de penicilina presente en la vacuna no ha causado complicaciones. Después del proceso de desnaturalización por formalina queda muy poca penicilina.

c. Efectos del tejido del virus del mono:
Cantidad de O.2 mg de proteina viral por mililitro y 150 mg de proteina del riñón por mililitro de vacuna no producen efecto alguno, según la evidencia reportada en los estudios de Mayer y por los contajes de Addis de niños vacunados.
d. No se ha reportado producción de anticuerpos incompatibles de tipo Rh o Hr en personas vacunadas.

11. **¿Qué hay del efecto provocativo de la vacuna?**

a. Los estudios de Bodian indican que el riesgo no es de importancia. Es aparente que ciertos procedimientos pueden ser provocativos si se aplican durante el período de la viremia, tal como inyecciones de penicilina y vacuna de pertussis. Esto no ocurre con la Vacuna Salk.
b. Demostrado en la epidemia de Hawaii en personal del Ejército y su control con la vacuna de polio.

12. **¿Deben vacunarse los pacientes que han enfermado clínicamente con poliomielitis paralítica o no paralítica?**

a. Sí, ya que pueden surgir segundos ataques debido a otros tipos de virus.

13. **Otros problemas prácticos en la administración de la vacuna:**

a. El pH de la vacuna es de 7.4 a 7.6.
El color rosa se debe al indicador fenol rojo que contiene.
b. Puede administrarse intradermalmente en dosis de 0.1 ml equivalente a 1 ml intramuscular. Se continúa recomendando la ruta intramuscular y en Puerto Rico en el brazo izquierdo.
c. El problema del Mertiolato (1:10,000) en la vacuna ha sido resuelto con la adición de Versen que remueve el Mertiolato.
d. Los frascos en uso al presente tienen una fecha de expiración a 5 meses en temperatura uniforme de 35º a 50ºF.

14. **¿Qué posibilidades hay de una vacuna segura de virus vivo?**

a. No hay ninguna al presente que pueda usarse o que el gobierno autorice.
b. Dos investigadores al presente trabajan con una vacuna de virus vivo atenuado:
Dr. Kopronski, Laboratorios Lederle
Dr. Albert D. Sabin, Cincinnati

15. **¿Puede una comunidad vacunada o un individuo que ha tenido buena respuesta de anticuerpos al inmunizarse, reinfectarse si se expone a la infección?**

a. Mientras el grado de anticuerpos esté a determinado nivel (1:100) no puede reinfectarse — no desarrollan la infección del canal alimenticio ni se elevan los niveles de anticuerpos.
b. Si los niveles de anticuerpos bajan de 1:100 la persona desarrolla la infección del canal alimenticio, elimina virus de la excreta y el titraje de anticuerpos se eleva.
c. En la comunidad, el niño que ha sido vacunado y ha desarrollado anticuerpos demostrará una fluctuación en el titraje de éstos, como sigue:
   1) Poco después de vacunarse será elevado.
   2) Estos niveles se reducirán poco a poco.
   3) Recibirá una dosis de reactivación.
   4) Volverá a elevarse el nivel de anticuerpos y luego quizás volverá a reducirse.
   5) Es posible que en cierto momento estos individuos tengan niveles relativamente bajos de anticuerpos, y entonces, estarán susceptibles a infección si son expuestos.
d. La reinfección en individuos parcialmente inmunizados se demuestra con un período más corto de eliminación de virus en la excreta que durante la infección primaria y la cantidad de virus eliminada es menor.
e. Los virus se encuentran en países donde no hay polio clínicamente diagnosticable. Ejemplo: En Egipto, Filipinas. Donde hay la mayor cantidad de virus hay menos parálisis.

16. **¿Qué hay sobre el futuro de la manufactura de la vacuna de polio?**

a. La vacuna tiene una efectividad consistente y uniforme.
b. La potencia de la vacuna en uso al presente es satisfactoria —ésta ha sido probada por el doctor Salk.
c. La potencia de la vacuna del 1955 es igual a la del 1954.
d. La meta es vacunar a toda la población hasta los 40 - 45 años con 2 dosis de vacuna — 120 millones de habitantes.

17. **¿Cómo es la respuesta inmuno-génica del individuo vacunado?**

a. Mayor lapso de tiempo entre la primera y segunda dosis resulta en una mejor respuesta inmunológica.

b. Ochenta por ciento (80%) de los individuos que no tenían anticuerpos detectables, a las 2 semanas demuestran éstos. Con la segunda dosis se cambian los 20 que no tenían anticuerpos detectables a detectables—casi el 100%.

c. Un lapso de 4, 6, 8, 9 ó 10 semanas es mejor.

18. **¿Cuándo no se debe aplicar la vacuna?**

a. En una familia donde se acaba de diagnosticar un caso de polio, porque sabemos que al tiempo de hacer el diagnóstico práctícamente todo niño en esa familia que no es inmune está infectado y puede estar en la fase de la viremia. No se debe aplicar la vacuna o la Gamma Globulina porque no hacen nada, y es muy tarde.

19. **¿Qué efectividad protectora tiene la vacuna al presente?**

a. 80%.

b. Con la vacunación del 1954 y 1955 se evitaron de 1200—1500 casos y de 60 — 70 muertes.

20. **¿Cuando la madre embarazada ha recibido la vacuna Salk, a qué grado está protegido el recién nacido?**

a. No se sabe.

21. **¿Debe aplicarse la vacuna en presencia de una infección de las vías respiratorias o conjuntamente con otras clases de vacunas?**

a. Sí.

b. Puede vacunarse contra el polio y viruela a la misma vez, pero no en el mismo brazo.

22. **¿Se debe vacunar un contacto?**
Sí, debe vacunarse y recibir Gamma Globulina (Contacto según se explica en el Núm. 12, de la sección Poliomielitis del presente artículo.)

23. **¿Está aumentando el número de casos en la población adulta?**

Sí, esa tendencia ya ha sido observada en Estados Unidos y y en Puerto Rico.

24. ¿Qué efecto tiene la tercera dosis?

Eleva el nivel de anticuerpos por encima de los niveles producidos por la primera y segunda inyección. Por lo tanto, completa la inmunización del individuo. El nivel de anticuerpos ha ido declinando cuando recibe la tercera dosis.

25. ¿Habrá que inyectar los niños todos los años para asegurar su protección?

Probablemente no, pues los que hace tres años se vacunaron aún tienen un nivel persistente elevado de anticuerpos.

26. ¿Qué data estadística tenemos al presente sobre la vacunación en masa?

a. Vacunados en los EE.UU. por lo menos una vez en el 1954-1955 (por lo general menores de 7 y 8 años) __ 7 millones
b. Vacunados en Puerto Rico por lo menos con la primera dosis ________________________________ 430,828
c. Total de dosis aplicadas en Puerto Rico ____ 900,562

27. Contraindicaciones revisadas —

a. Enfermedad aguda severa.
d. Durante la estación de verano (estación de polio) o durante epidemias de polio, a personas que demuestran síntomas de leve enfermedad, especialmente fiebre, dolor de garganta o condición gastrointestinal.
c. Contraindicación a todo producto biológico:
En ningún hogar donde haya un caso de polio se debe vacunar.

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659 Unión - Miramar
San Juan, Puerto Rico

INFORME DEL SECRETARIO

— Año 1956 —

Señores Miembros de la Cámara:

Pláceme someter a la consideración de ustedes la siguiente información:

Estadística Médica

Al 30 de noviembre los records a nuestra disposición arrojan las siguientes cifras en relación con el número de médicos registrados en nuestra Isla. Número total de médicos, 1510, cuya distribución por distritos es como sigue:

TABLA I

DISTRIBUCION DE MEDICOS POR DISTRITOS

Area	Socios	No Socios	Internos y Residentes	Licencia Especial	Total
Distrito Este	506	194	126	35	861
Distrito Norte	46	72	35	18	171
Distrito Sur	79	34	44	20	177
Distrito Oeste	44	16	1	14	75
	675	316	206	87	1284
Fuera de Puerto Rico					
Estudiando	35	36			71
Fuerzas Armadas	27	54			81
Trabajando	7				7
Subtotal	744	406	206	87	1443
Direcciones desconocidas					67
Total médicos registrados					1510

Descomponiendo aún más nuestros records encontramos que la distribución por pueblos es como sigue:

TABLA II

DISTRIBUCION DE MEDICOS POR PUEBLOS

DISTRITO NORTE

Pueblo	Socios	No Socios	Internos y Residentes	Licencia Especial	Total
ARECIBO	17	29	21	4	71
AGUADILLA	8	12	14	3	37
BARCELONETA	1	0	0	1	2
CAMUY	1	2	0	0	3
CIALES	1	2	0	0	3
COROZAL	1	0	0	1	2
DORADO	0	1	0	2	3
HATILLO	0	3	0	0	3
ISABELA	1	1	0	2	4
LARES	0	2	0	1	3
MANATI	6	1	0	1	8
MOROVIS	0	2	0	1	3
QUEBRADILLAS	2	0	0	0	2
SAN SEBASTIAN	3	4	0	1	8
TOA ALTA	0	1	0	0	1
TOA BAJA	0	2	0	0	2
UTUADO	2	6	0	0	8
VEGA ALTA	2	0	0	0	2
VEGA BAJA	1	4	0	1	6
TOTALES	46	72	35	18	171

DISTRITO OESTE

Pueblo	Socios	No Socios	Internos y Residentes	Licencia Especial	Total
MAYAGUEZ	30	9	0	4	43
AGUADA	1	1	0	0	2
AÑASCO	0	0	0	1	1
CABO ROJO	3	1	0	0	4
HORMIGUEROS	0	0	0	0	0
LAJAS	2	0	0	2	4
LAS MARIAS	0	1	0	1	2
MARICAO	0	0	0	2	2
MOCA	0	0	0	2	2
RINCON	0	1	0	2	3
SABANA GRANDE	2	1	0	0	3
SAN GERMAN	6	2	1	0	9
TOTALES	44	16	1	14	75

DISTRITO ESTE

Pueblo	Socios	No Socios	Internos y Residentes	Licencia Especial	Total
SAN JUAN	314	89	62	5	470
AGUAS BUENAS	1	0	0	0	1
BARRANQUITAS	0	1	0	1	2
BAYAMON	24	23	24	4	75
CAGUAS	15	9	0	2	26
CATAÑO	1	2	0	0	3
CAROLINA	2	3	0	1	6
CANOVANAS	0	2	0	2	4
CAYEY	6	3	0	2	11
CEIBA	0	0	0	0	0
COMERIO	1	0	0	0	1
CULEBRA	0	0	0	0	0
CIDRA	0	2	0	1	3
FAJARDO	9	3	22	0	34
GUAYNABO	0	1	0	2	3
GURABO	1	0	0	0	1
HUMACAO	10	3	0	0	13
JUNCOS	3	1	0	0	4
LAS PIEDRAS	0	1	0	1	2
LUQUILLO	0	1	0	2	3
NAGUABO	1	0	0	1	2
NARANJITO	0	1	0	0	1
RIO GRANDE	1	2	0	1	4
RIO PIEDRAS	114	43	18	8	183
SAN LORENZO	1	0	0	1	2
TRUJILLO ALTO	0	2	0	0	2
VIEQUES	0	2	0	0	2
YABUCOA	1	0	0	1	2
ST. THOMAS	1	0	0	0	1
TOTALES	506	194	126	35	861

DISTRITO SUR

Pueblo	Socios	No Socios	Internos y Residentes	Licencia Especial	Total
PONCE	57	11	44	6	118
AIBONITO	0	4	0	2	6
ARROYO	2	1	0	2	5
ADJUNTAS	0	2	0	3	5
COAMO	2	0	0	1	3
GUANICA	1	1	0	0	2
GUAYAMA	7	4	0	2	13
GUAYANILLA	1	0	0	0	1
JAYUYA	0	1	0	1	2
JUANA DIAZ	4	0	0	1	5
MAUNABO	0	1	0	0	1
PATILLAS	0	1	0	0	1
PEÑUELAS	0	0	0	1	1
OROCOVIS	1	1	0	0	2
SALINAS	1	2	0	0	3
SANTA ISABEL	0	1	0	0	1
VILLALBA	0	2	0	0	2
YAUCO	3	2	0	1	6
TOTALES	79	34	44	20	177

De los datos anteriormente transcritos se desprende que la matrícula de nuestra Asociación está integrada actualmente por 744 miembros. Sin embargo, 62 de éstos se encuentran temporalmente fuera del país, 35 cursando estudios avanzados y 27 sirviendo en las fuerzas armadas. Tenemos también 7 compañeros que han establecido su práctica en los Estados Unidos que han continuado siendo miembros de nuestra agrupación. En la siguiente tabla damos una relación de estos compañeros:

TABLA III

MEDICOS FUERA DE PUERTO RICO — ESTUDIANDO

1. Alemañy, Carlos E.
2. Almodóvar, Ramón I.
3. Armstrong, Carlos
4. Arroyo Jiménez, Pedro
5. Bajandas, Francisco
6. Bond, Walter J.
7. Chiqués, Carlos M.
8. Colón Fontán, Angel B.
9. Díaz, Angeles
10. Durand, Pedro J.
11. Fagot Rodríguez, Gabriel
12. Fuentes, Claude
13. García Ramírez, Oscar
14. Garriga, José
15. García Blanco, José
16. Gómez, Hirám E
17. Hidalgo Cestero, Carlos
18. Jiménez Vélez, José Luis
19. King, Robert R.
20. Limeres, José R.
21. Maestre, Federico J.
22. Mella, José Luis
23. Monserrate Anselmi, F.
24. Morales, Carlos E.
25. Molinary, José G.
25. Mussenden, Borinquen
27. Nine Curt, José
28. Navarro, Félix A.
29. Parés Dávila, María Amalia
30. Rodríguez García, A.
31. Sánchez Longo, Luis
32. Toro Nazario, Rafael
33. Tulla, Miguel A.
34. Torres, Zahidée M.
35. Vázquez Vélez, José A.

EN LAS FUERZAS ARMADAS

1. Alvarez de Choudens, José
2. Aybar, José A.
3. Axtmayer, Robert W.
4. Bierley, John R.
5. Betances Campora, José
6. Buxeda, Fernando
7. Domínguez, Alberto M.
8. Fernández Sariego, G.
9. Garrido Carmona, Manuel
10. Glover, Samuel I.
11. Irizarry Bulls, Edgard
12. Isales, Luis M.
13. Jiménez Pabón, Edwin
14. Márquez, Iván
15. Olmedo, José C.
16. Ortiz Gordils, Edgardo
17. Pérez Lara, Rafael
18. Ramos González, Luis
19. Rivera Cintrón, F. J.
20. Rengel, Ricardo
21. Rubio, Luis A.
22. Sifontes, José E.
23. Rivera Trujillo, Antonio
24. Toro, Russell A. del
25. Torres Machín, Arturo
26. Vallés, Héctor M.
27. Yumet, Angel M.

TRABAJANDO

1. Acosta Velarde, Antonio
2. Aguayo, Roberto
3. Domínguez, Carlos
4. Hernández, Rafael
5. Janer, Manuel
6. Ramos, Raúl L.
7. Sugrañes, José G.

Nuevos Miembros

Durante el año que finaliza se han acogido a nuestras filas los siguientes compañeros:

MIEMBROS REGULARES

1. Alvarez, Rafael
2. Báez, Ignacio José
3. Banuchi, Iván B.
4. Bendeck, Taufick Elías
5. Burden, Kenneth Hudson
6. Carlo Aymat, Enrique
7. Colberg Pujadas, Pedro Nelson
8. Fernández, Rafael O.
9. Fernández Durán, Manuel
10. Flores Gallardo, Arturo
11. Galindo, Lorenzo
12. García Ramírez, Iván H.
13. Grana Rodríguez, Julieta
14. Iguina Mora, Martín
15. Jiménez Mercado, Juan F.
16. Juan Jiménez, Abel F. de
17. Maynardi Reyna, Luis E.
18. Mari Rodríguez, Paul
19. Martínez Martínez, Isidro
20. Matta Méndez, Carlos R.
21. Mattos Nieves, Angel M.
22. Morris, Oriel C.
23. Noguera, Juan A.
24. Ortiz Quiñones, Julio A.
25. Otero, Pedro Jaime
26. Pagán Carle, Josué
27. Pagán, Víctor J.
28. Pendleton, Adaline
29. Rivera Rivera, Edwin
30. Rodríguez, Roberto C.
31. Rosa Febles, César A.
32. Ruiz Cruz, Ismael
33. Ruiz Sosa, Oscar
34. Sáez Fontany, Florencio
35. Serrano de Solis, Rosa A.
36. Somohano Mosquera, Angel M.
37. Tamm, Arkadi
38. Tapp, Jesse Washington
39. Toro Pérez, Jaime
40. Vargas Huerta, Nicolás
41. Vázquez San Martín, José
42. Vicéns, Enrique A.
43. Vilar, Ismael
44. Vilella Lecaroz, Juan José

MIEMBROS AFILIADOS

1. González Flores, Bernardino
2. Guerrero Guerrero, Rafael
3. Martínez Cancel, Zenobio R.
4. Nieves López, Pedro Luis
5. Pagán Luna, Victoriano
6. Shepard, Jack
7. Vázquez Casanova, José O.
8. Viñas Sorbá, Luis A.

A la fecha en que redactamos este informe están pendientes de consideración las solicitudes de los siguientes colegas:

1. Romero Gelpí, Angel Plinio
2. Olmo, Jaime Alberto (M.A.)
3. Silva, Jr., Armando
4. Weissenberg, Eugene

Bajas

Las bajas registradas este año han sido las siguientes:

Por Muerte

Dr. J. E. Luigi
Dr. Mario Juliá
Dr. Jeramfel Cordero
Dr. Alfredo V. Bou
Dr. Ramón C. Umpierre
Dr. Víctor Gutiérrez Ortiz

Por haberse ausentado del país

Dr. Alberto Adam
Dr. Juan R. Cabrera
Dr. Celso Ramón García
Dr. Donald F. Gowe
Dr. Antonio E. Molina
Dra. Lydia Pérez Guardiola

Por no pagar sus cuotas

Dr. Nicolás Méndez Hernández
Dra. Edith Z. Rodríguez
Dr. Nicolás Sanabria
Dr. Luis A. Sánchez
Dr. Federico Trilla

Reuniones Administrativas

Estamos incluyendo como parte de este informe una relación de las reuniones de carácter administrativo celebradas por los diferentes organismos de la Asociación durante el año que termina. Si tenemos en cuenta que este año se han celebrado 123 reuniones de carácter administrativo nos daremos cuenta de la magna labor realizada durante este período. Entre los organismos que con más regularidad se han reunido se destacan los siguientes:

Junta de Directores
Comité de Relaciones Públicas
Comité de Credenciales
Comité Científico
Junta Editora del Boletín
Comité de Querellas
Junta del Auxilio Médico Mutuo

Reuniones Científicas

Si grande ha sido la labor realizada por la Asociación en su aspecto administrativo no lo ha sido menos en el aspecto científico. Se han celebrado este año 55 actos de carácter científico, habiéndose presentado un total de 105 conferencias. Véase la relación de los temas discutidos al final de este informe.

Asociación Médica Americana

A la fecha de este informe 200 compañeros han pagado su cuota a la Asociación Médica Americana y 13 son miembros exen-

tos del pago de cuota, lo que hace un total de 213. Aunque esta cifra es mucho mejor que la del año pasado, consideramos que la misma puede y debe ampliarse.

Los servicios que presta la A.M.A. a nuestra agrupación en general, y la ayuda y atenciones que reciben nuestros directores cuando tienen que acudir a Washington o a Chicago en gestiones oficiales deben tenerse en cuenta por cada uno de nuestros socios y nuestra meta debe ser la de hacer que cada médico asociado sea al mismo tiempo miembro de la Asociación Médica Americana.

Incluímos al final del presente informe una relación de los compañeros que han pagado su cuota por nuestro conducto este año.

Actividades generales

Hemos hecho cuanto ha estado a nuestro alcance para cumplir con los deberes que nos fueron impuestos. Acompañamos al Sr. Presidente en uno de sus viajes a Chicago; hemos atendido a la correspondencia que vino a nuestro cargo; se han expedido las certificaciones correspondientes a los compañeros que las solicitaron, hemos remitido a la Asociación Médica Americana toda la información que se nos ha solicitado y hemos formado parte del Comité Pro Seguridad de Tránsito.

Cordialmente,

RAFAEL A. GIL, M.D.
Secretario

RELACION DE REUNIONES ADMINISTRATIVAS CELEBRADAS DURANTE EL AÑO 1956

Fecha		*Organismo*	*Núm. de Asistentes*
1955			
Diciembre	17	Junta de Directores	7
1956			
Enero	3	Comité de Relaciones Públicas	6
	28	Junta de Directores	6
	31	Junta Editora del Boletín	5
	31	Comité de Relaciones Públicas	4
Febrero	7	Comité Científico	6
	7	Junta de Directores, Comité Escudo Azul	

Fecha		*Organismo*	*Núm. de Asistentes*
		y Cruz Azul	9
	11	Directiva Distrito Este	5
	14	Comité de Relaciones Públicas	6
	17	Junta Editora del Boletín	4
Marzo	6	Comité de Relaciones Públicas	6
	13	Comité de Relaciones Públicas	5
	14	Comité de Credenciales	4
	15	Junta de Directores	6
	16	Junta Editora del Boletín	4
	19	Comité de Damas Auxiliares	10
	20	Comité Dr. Costa Mandry, Forastieri y Licha	3
	20	Comité de Relaciones Públicas	6
	20	Comité Dr. Hernández Morales	3
	27	Comité de Relaciones Públicas	3
	21	Junta de Directores, Asoc. Méd. Dtto. Este y Comité Científico	10
Abril	3	Comité de Relaciones Públicas	5
	6	Comité de Damas Auxiliares	12
	10	Comité de Relaciones Públicas	5
	13	Comité Científico Dtto. Este	4
	14	Directiva y Comité Seguros Voluntarios con Sr. Castellucci	11
	14	Cámara de Delegados	41
	19	Comité Científico	5
	24	Comité de Relaciones Públicas	6
	26	Directiva Dtto. Este	5
	26	Comité de Credenciales	4
	28	Junta de Directores	10
Mayo	1	Comité de Relaciones Públicas	7
	3	Comité de Drogas	3
Mayo	4	Junta Editora del Boletín	4
	11	Comité de Querellas	3
	15	Comité de Relaciones Públicas	8
	17	Comité de Cáncer	6
	17	Comité Científico	5
	22	Comité de Relaciones Públicas	8
	25	Junta Editora del Boletín	4
	28	Comité Científico Dtto. Este	4
	29	Comité de Relaciones Públicas	6
	31	Comité de Credenciales	4

Fecha		*Organismo*	*Núm. de Asistentes*
	31	Directiva Dtto. Este	6
	31	Comité de Malpractice	3
Junio	2	Junta de Directores	7
	5	Comité Práctica de la Medicina	2
	6	Comité de Relaciones Públicas	9
	12	Comité de Querellas	4
	12	Comité de Relaciones Públicas	9
	14	Comité de Credenciales	4
	18	Comité de Relaciones Médicos y Hospitales	3
	19	Comité de Relaciones Públicas	6
	22	Directiva médicos Práctica General	6
	26	Comité de Relaciones Públicas	3
Julio	3	Comité de Relaciones Públicas	4
	7	Junta de Directores	8
	9	Comité de Práctica Médica	2
	10	Comité de Credenciales	3
	11	Comité de Relaciones Públicas	19
	16	Comité de Práctica Médica	3
	18	Junta de Directores	9
	19	Comité Científico	5
	20	Comité Relaciones Médicos y Hospitales	4
	21	Junta de Directores	10
	24	Comité de Credenciales	4
	24	Comité de Relaciones Públicas	6
	24	Comité de Escudo Azul	5
	26	Comité de Querellas	4
	31	Comité de Relaciones Públicas	5
Agosto	4	Junta de Directores	10
	7	Comité de Relaciones Públicas	8
	7	Directiva Dtto. Este	5
	11	Cámara de Delegados	27
	14	Comité de Relaciones Públicas	3
Agosto	14	Comité de Mediación y Querellas	4
	19	Directiva y funcionarios Escuela Médica	13
	21	Comité de Mediación y Querellas	4
	21	Comité de Relaciones Públicas	8
	22	Comité de Cáncer	6
	23	Comité de Damas Auxiliares	4
	28	Comité de Relaciones Públicas	4
	28	Comité Práctica Privada y Directiva	6

Fecha		*Organismo*	*Num. de Asistentes*
Septiembre	1	Directiva y Presidentes Secciones	9
	4	Comité de Relaciones Públicas	6
	4	Directiva Dtto. Este	7
	6	Comité de Credenciales	4
	11	Comité de Relaciones Públicas	6
	18	Comité de Relaciones Públicas	2
	22	Junta de Directores	5
	25	Comité de Relaciones Públicas	4
	25	Comité de Damas Auxiliares	12
	27	Comité Científico	5
	27	Comité de Cáncer	6
	28	Junta Editora del Boletín	4
Octubre	2	Junta de Directores	6
	2	Comité de Relaciones Públicas	3
	9	Comité de Relaciones Públicas	4
	17	Comité Científico	4
	19	Junta Editora del Boletín	5
	20	Junta de Directores	6
	23	Comité de Relaciones Públicas	4
	24	Comité de Convención	3
	30	Comité de Relaciones Públicas	5
Noviembre	8	Comité de Malpractice	4
	15	Comité de Cáncer	6
	16	Asamblea Sección Médicos Generalistas	12
	20	Comité de Nominaciones	5
	21	Comité de Credenciales	4
	23	Comité de Convención	2
	24	Junta de Directores	8
	5	Comité Relaciones Públicas	4
	12	Comité Relaciones Públicas	4
	20	Comité Relaciones Públicas	5
	27	Comité Relaciones Públicas	7

RELACION DE CONFERENCIAS CIENTIFICAS CELEBRADAS DURANTE EL AÑO 1956

Fecha		*Tema y Autor*	*Auspiciada por*
Diciembre	16	Posibles factores patológicos del cáncer del cuello del útero, Dr. Jesús García Orcoyen	Sección de Obstetricia y Ginecología
Enero	16	Lupus Erythematosus Disseminata, Dr. David N. Gould	Sección de Radiología
	17	Diagnostic Uses of Chlografin, Dr. David N. Gould	" " "
	19	Angiography in Surgically Amenable Congenital Heart Lesions, Dr. David N. Gould	" " "
	20	Arthritis and Arthrosis, Dr. David N. Gould	" " "
	31	Disquinesia biliar, Dr. C. González Bueno	Curso Postgraduado
		La evolución cancerosa de la vesícula litiásica, Dr. H. G. Mogena	" "
Febrero	1	Algunas sombras raras en la radiología del abdomen, Dr. Gutiérrez Arrese	" "
		Fístulas internas y externas de los operados gástricos, Dr. C. González Bueno	" "
	2	Colitis ulcerosa no específica, Dr. Gutiérrez Arrese	" "
		Clínica de las Pancreopatías, Dr. H. G. Mogena	" "
	27	The Problem of Drug Allergy, Dr. Richard A. Kern	" "
	28	The Allergy in Cardiovascular Disease, Dr. Richard A. Kern	" "
	29	Uses and Abuses of Steroid Therapy, Dr. Richard A. Kern	" "
Marzo	1	The Medical Challenge of the Forties, Dr. Richard A. Kern	" "
	2	Rehabilitation in Geriatrics, Dr. Richard A. Kern	" "
	11	1. Symposium on Adrenocortical Steroids	Asoc. Médica Dtto. Oeste
		a) Introduction, Dr. A. M. de Andino, Jr.	" " " "
		b) Physiological Aspects of Aldosterone, the Adrenal Salt Hormone, Agustín M. de Andino, Jr., M.D.	" " " "
		c) The Newer Synthetic Adrenal Steroids, Dr. Manuel E. Paniagua	" " " "

Fecha	*Tema y Autor*	*Auspiciada por*
	d) Use and abuses of Cortisone and its Derivatives, José A. de Jesús, M.D.	Asoc. Médica Dtto. Oeste
	e) Discussion	" " " "
"	2. Employment and Limitations of Roentgenologic Procedures in Destructive Pulmonary Lesions, Dr. M. Guzmán Rodríguez and Carlos Guzmán Acosta, M.D.	" " " "
	3. Clinicopathological Conference - Surgical Case, Dr. Donald Jutzy	" " " "
14	Present Day Techniques in Psychiatry, Dr. David J. Impastato	Sección de Psiquiatría y Neurología
	Constructive Relationship between Faith and Psychiatry, Dr. Ferruccio Di Cori	" " "
22	Hepatic Insufficiency, encephalopathy and coma with special reference to treatment, Dr. Cecil J. Watson	Curso Postgraduado
23	The porphyrins and porphyria; recent advances in fundamental and clinical aspects, Dr. Cecil J. Watson	Curso Postgraduado
24	The problem of jaundice with remarks on diagnosis and treatment, Dr. Cecil J. Watson	Junta Cursos Postgraduados
26	Hepatic cirrhosis; classification prognosis and treatment, Dr. Cecil J. Watson	" " "
27	The rational treatment of the anemias, Dr. Cecil J. Watson	
Abril 17	Medical Problems of Interest to Urologists and Gynecologists, Dr. Luis A. Sanjurjo	Sección de Obstetricia y Ginecología
17	Actividades de relaciones públicas desarrolladas por la Asociación Médica Americana y las Asociaciones Médicas de Estados Unidos, Sr. Leo E. Brown	Comité de Relaciones Públicas de la A.M.P.R.
Mayo 9	Dental Problems in Pediatrics, Dr. Marcos A. Dones	Sección de Pediatría
15	Problemas del Recién-Nacido	Sección de Obstetricia y Ginecología
21	Celiac Disease and Cystic Fibrosis of the Pancreas, Dr. Milton Rapoport	Curso Postgraduado
22	Inborn Errors of Metabolism, Dr. Milton Rapoport	" "

Fecha		*Tema y Autor*	*Auspiciada por*
	23	Hypoglycemia and Disordered Carbohydrate Metabolism in Childhood, Dr. Milton Rapoport	Curso Postgraduado
	24	The Management of the Child with Renal Failure, Dr. Milton Rapoport	" "
	25	Growth Failure in Childhood, Dr. Milton Rapoport	" "
Junio	25	Contribución del Laboratorio al Diagnóstico Diferencial de la Ictericia, Dr. Héctor Ducci	Sección de Gastroenterología
	26	Tratamiento de la Hepatitis Aguda con la Cortisona, Dr. Héctor Ducci	" " "
	26	Present Status of the Treatment of Carcinoma of the Cervix, Dr. Juan del Regato	Sección de Radiología, Comité de Cáncer, y el Negociado de Cáncer
	27	Hepatitis post-transfusional, Dr. Héctor Ducci	Sección de Gastroenterología
Julio	19	Research and Treatment in Alcoholism, Dr. Israel Zwerling	Sección de Psiquiatría y Neurología
Agosto	11	Common Nasal Conditions and their Management, Dr. Enrique Vicéns	Asoc. Médica. Dtto. Sur
		Hypothyroidism in Children, Drs. Catalina Scarano and Jenaro Scarano	" " " "
		Circumscribed Lesions of the Chest, Dr. R. A. Pérez Ribié	" " " "
		The Management of Strabismus in Children, Dr. José Fiol Bigas	" " " "
	12	Thrombotic Thrombocytopenic Purpura, Drs. Héctor Rodríguez, Donald Babb and Jaime Costas Durieux	" " " "
	12	Chondrodystrophies, Dr. José Dávila López	" " " "
		Indicaciones de Resección en Tuberculosis pulmonar, Dr. Jaime Costas Durieux	" " " "
		Clinicopathological Conference, Dr. Joseph Brinz and Edwin Rivera	" " " "
	23	Otitis Media en Pediatría (película)	Sección de Pediatría
	24	Panel sobre Esquistosomiasis	
		1. Introducción, Dr. Rafael Rodríguez Molina	Asoc. Médica. Dtto. Este

Fecha	Tema y Autor	Auspiciada por
	2. Aspecto Inmunológico, Col. David H. Naimark	Asoc. Médica Dtto. Este
	3. Aspecto Clínico	" " " "
	a) Hepato-esplénico, Dr. Héctor E. Rodríguez	
	b) Pulmonar, Dr. E. J. Marchand	
	4. Aspecto Quirúrgico	" " " "
	a) Dr. Francisco L. Raffucci	
	b) Dr. D. Rodríguez Pérez	
	5. Discusión y Preguntas	" " " "
	6. Resumen, Dr. R. Rodríguez Molina	" " " "
25	The Practical Use of X-Ray Procedures in Obstetrics, Dr. Edward O'Neill	" " " "
	La poliomielitis y la Vacuna Salk, Dr. Enrique Matta, Jr.	" " " "
	Conferencia clínico-patológica Dr. F. Hernández Morales, Moderador	" " " "
	a) Presentación Radiológica Dr. R. Díaz Bonnet	" " " "
	b) Discusión Clínica Dr. Ramón A. Sifre y Dr. Calixto A. Romero	" " " "
	c) Discusión y Preguntas	" " " "
	d) Discusión patológica Dr. R. Ramírez Weiser	" " " "
	e) Comentarios finales Dr. F. Hernández Morales	" " " "
Septiembre 6	Diagnostic Approach to Behavior Problems, Dr. Harry Bakwin	Sección de Pediatría
	The Management of Behavior Disorders in Children, Dr. Harry Bakwin	" " "
7	The Academy and the Puerto Rico Medical Association, Dr. Jaime F. Pou	" " "
	The Academy and the Dept. of Health, Dr. Juan A. Pons	" " "
	The Academy and the Medical School, Dr. E. Harold Hinman	" " "
	The Academy and its Functions, Dr. Harry Bakwin	" " "
Septiembre 20	Carcinoma del Seno, Dr. Eugenio M. de Hostos	Sección de Cirugía General
21	Ocular Biomicroscopy (Películas) " Bacteriology	Sección de Oftalmología y Otorrino.

Fecha		*Tema y Autor*	*Auspiciada por*
	30	Lesiones Ilio-cólicas con obstrucción, Dr. Raúl A. Marcial Rojas	Asoc. Médica Dtto. Norte
		Afibrinogenemia en los estados de embarazo, Dr. J. Rodríguez Olmo	" " " "
		Hernias inguinales, Dr. Antonio H. Susoni	" " " "
		El valor de la retención urinaria en el diagnóstico diferencial de las enfermedades cardiovasculares, Dr. R. Arrillaga Torrens	" " " "
Octubre	1	Respiratory Tract Infections and their Treatment, Dr. Hobart A. Reimann	Junta Cursos Postgraduados
	2	Antibiotic Therapy, Dr. Hobart A. Reimann	" " "
	3	Presentation and Discussion of an Interesting Case, Dr. H. A. Reimann	" " "
	5	Recent Important Practical Advances in Infectious Diseases, Dr. Hobart A. Reimann	" " "
	6	Histoplasmosis, Dr. H. A. Reimann	" " "
	7	Clinico-pathological Conference, Dr. Enrique Koppisch and Dr. Hobart A. Reimann	" " "
	24	Tonsilectomía, Dr. José Picó	Sección de Pediatría
	26	Eosinophylic Granuloma of the Lung - Report of a Case, Dr. Laszlo Ehrlich	A.M.P.R. y American Coll. of Physicians
		Vicarious Bleeding, Report of a Case, Dr. F. Diez Rivas	" " "
		A Symposium on Acute and Chronic Pancreatitis 1. Physiologic Considerations, Dra. Carmen Busó de Casas 2. Diagnosis and Medical Treatment, Dr. Ramón A. Sifre 3. Radiologic Picture Dr. C. Guzmán Acosta 4. Surgical Treatment Dr. Francisco L. Raffucci	" " "
	27	Etiology of 1954-55 Puerto Rican Poliomyelitis Epidemic, Dr. David H. Naimark	
		A Comparison between Rectal Biopsy, Stool Examination, Skin Tests Circumoval Precipitin Reaction, and Sigmoidoscopy in	

Fecha	*Tema y Autor*	*Auspiciada por*
	Schistosomiasis Mansoni, Dr. F. Hernández Morales	
	Jejunal Biopsies in Sprue. Dr. Charles E. Butterworth	
	Studies on Infiltrative Eosinophilia, Dr. R. Díaz Rivera y Zoilo R. Sotomayor	
	Post Operative Myocardial Infarction, Dr. Dwight Santiago and José H. Rivera Maldonado	
	Studies on the Pathogenesis of the Anemia of Hypothyroidism, Dr. Roberto Busó, Sara Olavarrieta, B.S., and Ramón M. Suárez, Jr., M.D.	
	Cardiovascular Disease in Puerto Rico, Dr. Enrique Koppisch and H. Vázquez Milán, M.D.	
9	Concepto actual de las treponematosis - Importancia sanitaria y control de las mismas, José Gay Prieto, M.D.	Sec. Dermatología
20	Pathologic and Therapeutic Aspects of Malignant tumors of the uterus excluding the cervix, R. A. Marcial y R. J. McConnie	Sec. Obst. y Ginec.
28	Research for the Ideal Antibiotic, Gerard Van Halsema, M.D.	Comité Científico

INFORME DEL TESORERO

Año 1956

Sr. Presidente; señores Miembros de la Cámara:

Respetuosamente sometemos a vuestra consideración el siguiente informe en cuanto al desenvolvimiento económico de nuestra Asociación durante el año que terminó el 30 de noviembre pasado.

Para que ustedes tengan una mejor idea en cuanto a la situación económica de la Asociación hemos llevado nuestras operaciones al día último de noviembre. Sin embargo, siguiendo el acuerdo adoptado por esta Cámara en su reunión de diciembre de 1955, nuestros libros fueron cerrados oficialmente el 31 de octubre para que los señores auditores tuvieran tiempo para proceder a la intervención de nuestra contabilidad durante el mes de noviembre. Dicha intervención de cuentas se llevó a efecto y el informe de los señores auditores nos fué entregado el día 4 de los cursantes y forma parte del nuestro.

Otro acuerdo de la Cámara al cual dimos cumplimiento este año fué el de gestionar una fianza para el Secretario Ejecutivo.

Damos a continuación un breve resumen del movimiento habido en las principales cuentas de la Asociación:

Cuotas

Hasta el 30 de noviembre los ingresos habidos por concepto de cuotas de miembros han montado a $20,925.00, lo que representa un aumento de $1,209 sobre la cantidad cobrada el año pasado, y $925 más que la cifra por nosotros estimada, que había sido de $20,000.

Nuestro registro de socios este año ha alcanzado la cifra de 744. De éstos hay 69 que están fuera del país y sólo unos pocos de ellos han satisfecho su cuota. Los siguientes compañeros están exentos de pagar la parte de la cuota correspondiente a la Asociación:

1. Alúm Pérez, José
2. Belaval, José S.
3. Berríos, Manuel B.
4. Bird, Jorge
5. Dunscombe, Colby W.
6. García de Quevedo, Luis
7. González, Carlos
8. Janer, Fernando
9. Montalvo Guenard, Andrés
10. Santos Tió, Luis F.

Tenemos además los siguientes miembros en la categoría de afiliados:

1. Bosch, José E.
2. Bernart, William F.
3. Bladuell Ramos, Walter
4. Frank, Julio E.
5. González Flores, Bernardino
6. Guerrero, Rafael
7. Martínez, Zenobio R.
8. Nieves López, Pedro L.
9. Pagán Luna, Victoriano
10. Rigau, José M.
11. Rubio, Luis A.
12. Shepard, Jack
13. Viñas Sorbá, Luis A.
14. Vázquez Casanova, José

Al 30 de noviembre hay un total de 62 médicos que no han satisfecho la cuota. (Véase relación en la página 55). Cuando dichos compañeros satisfagan su cuota corresponderá a la Asociación la cantidad de $1,938. El balance de los $3,054 adeudados por este grupo irá a engrosar los fondos del Auxilio Médico Mutuo.

Nos vimos precisados a dar de baja a seis compañeros que debían dos o tres años de cuota, y quienes en ningún momento respondieron a nuestras reiteradas gestiones para que se pusieran al día en sus obligaciones.

Auxilio Médico Mutuo

En la cuenta del Auxilio hemos tenido ingresos este año por la cantidad de $12,675 por concepto de cuotas y $951.76 por intereses devengados por las cuentas de ahorros, lo que hace un total de ingresos de $13,626.76.

Hemos pagado las pólizas de los siguientes compañeros fenecidos:

Dr. J. E. Luigi
Dr. Mario Juliá
Dr. Jeramfel Cordero
Dr. Ramón C. Umpierre

Está pendiente de liquidación la póliza del doctor Alfredo V. Bou, cuyos beneficiarios no nos ha sido posible localizar.

A la fecha de este informe tenemos cuotas por cobrar montantes a $1,116.

El balance neto que arroja el Auxilio Médico este año, después de descontar la póliza pendiente de pago, es de $6,088.51, cuyo fondo pasa a engrosar las reservas de nuestro plan de seguro, el cual tiene ahora un balance neto de $61,325.61.

Boletín Médico

Hasta el momento en que redactamos estas líneas la última edición publicada es la correspondiente al mes de agosto.

El total de ingresos por concepto de anuncios ha sido de $7,461.57, de los cuales $1,129.99 corresponden a anuncios del año pasado y $6,331.50 a anuncios publicados en las 10 ediciones que han aparecido este año (noviembre, 1955 a agosto, 1956). Tenemos cuentas por cobrar por concepto de anuncios montantes a $929.50. Los egresos habidos con motivo de la impresión y distribución del Boletín han subido a $6,355.67, que restados al total de anuncios publicados en esas ediciones, que ha sido de $7,261.08, nos deja un beneficio de $905.41.

Vitrinas para exhibiciones permanentes

Las vitrinas para exhibiciones permanentes este año nos han producido un ingreso de $1,450, que es inferior al obtenido el año pasado en la cantidad de $450. Ello se debe a que varias firmas descontinuaron la cooperación que venían ofreciéndonos. La firma José F. Bonelli nos debe $180.00 por concepto de la vitrina que ocupa.

Cursos Postgraduados

Este año se celebraron cuatro cursos postgraduados habiéndose logrado cubrir los gastos y dejando un pequeño superavit para hacer frente a otros gastos misceláneos, según se desprende de la tabla siguiente:

Curso a cargo de	Ingresos	Egresos
Dres. González Bueno, Gutiérrez, Mogena y Cibeira	$ 900.00	$ 700.00
Dr. Richard A. Kern	600.00	638.50
Dr. Cecil J. Watson	1,070.00	958.30
Dr. Hobart A. Reimann	310.00	438.20
TOTALES	$2,880.00	$2,735.00
Superavit		145.00

Del curso ofrecido por el Dr. Reimann están por cobrar $200.00 del Departamento de Salud y $40.00 de la Administración de Veteranos, que unidos a los $145.00 del superavit anotado en la tabla anteriormente transcrita llevan el balance a nuestro favor a **$385.00.**

Además de las transacciones anteriormente anotadas, este año cobramos $300 que nos adeudaban el Departamento de Salud y la Administración de Veteranos al finalizar el año pasado por concepto de su contribución para el curso dictado por el doctor Blankenhorn, y procedimos a pagar al Sr. Crawford $220 por el curso de fotografía que dictara el año pasado para un grupo de colegas.

Fondo pro Edificio

Los donativos recibidos este año para el Fondo pro Edificio han montado solamente a $1,340, lo que unido a los intereses devengados por las cuentas de ahorros del edificio ($945.44) y el beneficio del sorteo celebrado el año pasado ($6,237.11) lleva el total de este fondo al 30 de noviembre del año en curso a $39.057.44.

Locales para exhibiciones durante la asamblea

Estamos acompañando a este informe una relación de todas las firmas que han contratado espacio para exhibición durante la asamblea anual. El total de ingresos por este concepto ascenderá este año a $6,100. De dicha suma ya hemos cobrado $1,400 y se nos adeuda $4,700.

Cuentas de Ahorros

Adjuntamos a este informe una relación de las cuentas de ahorros con que cuenta la Asociación en la actualidad. Tenemos 9 cuentas con un balance entre todas de $83,863.41. Los intereses devengados por estas cuentas durante el último semestre de 1955 y hasta junio 30 de 1956, montaron a $2,262.73.

Bonos

Este año redimimos cuatro de los bonos que tenía nuestra Asociación. Por no tener nuestra agrupación una autorización especial que otorga el gobierno federal a las entidades de carácter no pecuniario, en el cambio de dichos bonos se nos hizo un descuento en los intereses devengados, y de $1,040 que nos correspondía por dicho concepto sólo recibimos $728. Inmediatamente interpusimos la correspondiente apelación ante el Colector Federal de Rentas Internas en Puerto Rico; pero aún no ha sido resuelto nuestro caso.

De los bonos en nuestro poder actualmente, hay siete que vencieron en marzo de este año y los cuales no hemos cambiado esperando se resuelva nuestra apelación, y tratar de conseguir

el interés total en dicho grupo que representa una cantidad de $4,940.

El precio original de los bonos que tenemos en la actualidad asciende a $23,680. $780.00 de éstos corresponden a la Asociación y los restantes $22,900 pertenecen al Auxilio Médico Mutuo.

Club Médico

El Club de nuestra Asociación ha seguido desenvolviéndose más o menos en idéntica forma a los últimos años. Los ingresos habidos este año por concepto de ventas han montado a $3,165.24, más $69.14 de intereses devengados en la cuenta de ahorros. Los egresos que se han registrado han sumado $2,043.74, que descontados de las ventas dejan un beneficio neto de $1,121.50. Al 30 de noviembre hay cuentas por cobrar por la cantidad de $177.65, lo que lleva el beneficio de este año a $1,299.15.

Durante el año la directiva celebró dos ágapes, uno en honor del Sr. Leo E. Brown y otro para la entrega de los premios Madison, y además se sirvieron obsequios en otras reuniones de la directiva y varios comités de la Asociación. El consumo de bebidas en estos actos ha montado a la cantidad de $135.55, suma ésta que ha mermado el beneficio neto del Club.

Es bueno hacer constar, sin embargo, que dicho beneficio se ha logrado porque no hemos remunerado los servicios extras prestados por Sánchez y su esposa. A este efecto, propusimos en la reunión del Comité de Finanzas, y así fué aprobado, que de los fondos del Club Médico se asigne la cantidad de $60.00 mensuales para remunerar los servicios de las personas a cargo del Club.

El balance actual del Club es de $5,758.62 en caja, más cuentas por cobrar montantes a $177.65 y mercancía por valor de $404.19.

Gastos extraordinarios

Este año nuestra Asociación ha tenido gastos extraordinarios, entre los cuales podemos mencionar los siguientes:

Relaciones Públicas (Sueldo del Director, viaje a Chicago del presidente del Comité, comidas y anuncios)	$3,078.27
Viaje del Sr. Presidente y el Sr. Secretario a Chicago	584.54
Viaje del Sr. Presidente a Miami	155.77
Balance gastos viaje Dr. Colón Yordán a Chicago, 1955	145.88

Viaje a Washington para firmar contrato Medicare (Esta cantidad nos será reembolsada más tarde por el Departamento de Defensa)	444.00
Libro del Año publicado por El Imparcial	200.00
Coronas y Esquelas	348.56
Total desembolsos extraordinarios	$4,957.02

Gastos generales

Los demás gastos de la Asociación se han matenido más o menos dentro de los límites de las partidas en presupuesto.

Medicare

Para facilitar el funcionamiento del programa Medicare, nuestra directiva ha decidido separar de los fondos ordinarios de la Asociación la cantidad de $10,000 para hacer frente a los gastos del primer mes. Los gastos administrativos del programa serán reembolsados por el Departamento de Defensa a medida que le enviemos nuestras facturas; pero es bueno aclarar que esos $10,000 deberán permanecer asignados al fondo especial de Medicare todo el tiempo por el cual se prolongue dicho programa.

Balance al 30 de noviembre de 1956

Al final del informe incluímos una serie de estados demostrativos de las operaciones de tesorería este año.

Podemos resumir la situación económica de la Asociación, a la fecha de este informe, como sigue:

CUENTA DE LA ASOCIACION

Banco Popular de Puerto Rico	$35,094.04	
Bonos	780.00	
Crédito y Ahorro Ponceño (Ahorros)	16,200.62	
First Federal Savings (Ahorros)	31,448.80	$83.523.46

Nota: En este balance están incluídos los siguientes fondos especiales:

Sección de Medicina General	73.79
Asociación Médica del Distrito Este	$ 379.32

Comité de Damas Auxiliares	1,028.75	
Fondo Pro Edificio	39,057.44	
Fondo Pro Hospital González Martínez	2,257.50	
Sección de Cardiología	22.00	
Sección de Dermatología	278.80	
Sección de Pediatría	1,320.44	
Sorteo carro 1956	2,525.00	46,943.04
Balance Neto de la Asociación		$36,580.42

CUENTA DEL AUXILIO MEDICO MUTUO

Banco Popular de Puerto Rico	$ 7,903.47	
Bonos	22,900.00	
Crédito y Ahorro Ponceño (Ahorros)	21,707.20	
First Federal Savings (Ahorros)	10,314.94	$62,825.61
Menos: Póliza Dr. Bou - por pagar		1,500.00
Balance en Caja		$61,325.61

CUENTA DEL CLUB MEDICO

Banco Popular de Puerto Rico	$1,620.03	
Crédito y Ahorro Ponceño (Ahorros)	4,129.59	$5,7[illegible]8.62

Proyecto de Presupuesto

Incluímos como parte de este informe un proyecto de presupuesto para el año 1956, el cual ha sido revisado por el Comité de Finanzas. En dicho proyecto de presupuesto hacemos un análisis de los gastos habidos durante el año que termina.

Estamos sugiriendo, con la aprobación del Comité de Finanzas, los siguientes aumentos:

	Sueldo Actual	Sueldo Propuesto
Ayudante del Secretario Ejecutivo	$2,100.00	$2,400.00
Clerk auxiliar	900.00	1,080.00
Conserje-mensajero	900.00	1,080.00
Bibliotecaria - part time	720.00	900.00

También se incluye la siguiente partida de nueva creación:

Gastos de viaje, Delegado A.M.A. -------------- 400.00

Con cargo a la cuenta del Club Médico se autoriza asimismo la siguiente erogación:

Sueldo personal a cargo del Club -------------- 720.00

❖ ❖ ❖

Para todos cuantos nos han ayudado a poder cumplir cabalmente con nuestro cometido vaya nuestra sincera expresión de gratitud.

Respetuosamente,

C. José Ferraioli, M.D.
Tesorero

BOLETIN
DE LA
ASOCIACION MEDICA
DE
PUERTO RICO

Organo Oficial de la Asociación Médica de Puerto Rico

Enero - Diciembre, 1956
Vol. 49

San Juan
Imprenta Venezuela
1956

LISTA DE COLABORADORES AL VOLUMEN 48 DEL

BOLETIN DE LA ASOCIACION MEDICA DE PUERTO RICO

ANDINO, Agustín M.
APONTE, Joseph L.
AREAN, Víctor M.
BLANCO, Gumersindo
BUXEDA, Roberto
CASANOVA DIAZ, A. S.
CASTELLUCCI, John W.
CHAFEY, David H.
COSTA MANDRY, Oscar
COSTAS DURIEUX, J. L.
CURBELO, Pablo G.
DIEZ RIVAS, Federico
EHRLICH, Laszlo
FINN, William F.
FLAX, Herman J.
FORTUÑO, Roberto F.
FOX, Irving
GARCIA, Arnaldo J.
GARCIA DE LA NOCEDA, Hilda
JESUS, José A. de
KERN, Richard A.
MARCIAL ROJAS, Raúl
MARTINEZ RIVERA, E.
MATTA, Enrique L.
OLAVARRIETA, Sara T.
O'NEILL, Edward
PANIAGUA, Manuel E.
PASSALACQUA, Luis A.
PONS, Juan A.
POU, Jaime F.
QUINTERO, Ernesto
RAMIREZ RODRIGUEZ, Elí A.
RODRIGUEZ, Héctor F.
RODRIGUEZ LUCCA, B.
RODRIGUEZ MOLINA, Rafael
RODRIGUEZ OLLEROS, A.
RODRIGUEZ, R. W.
SHERMAN, Irving C.
SIFONTES, José E.
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WEISSENBERG, Eugene H.

INDICE AÑO 1956

INDICE DE MATERIAS

INDICE DE AUTORES

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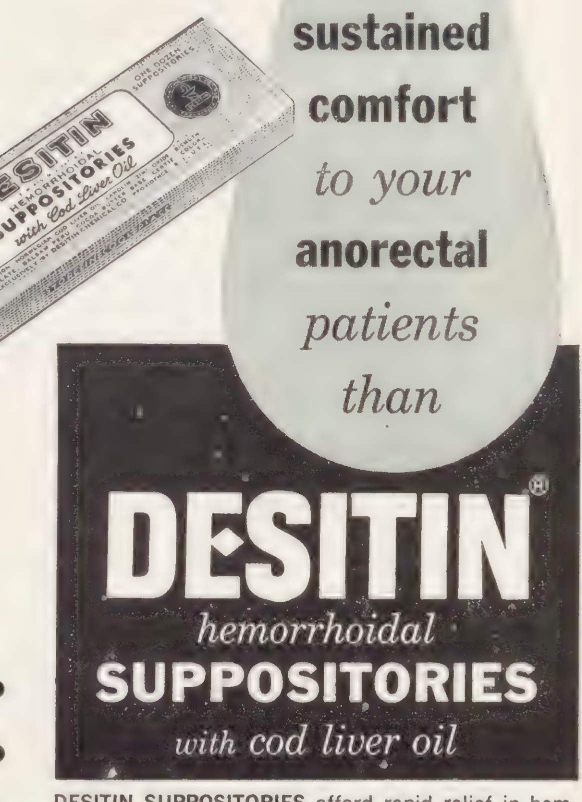


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